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SOME LABORATORY FINDINGS PERTAINING TO THE CLINICAL USE OF PENICILLIN.[1]

By SYDNEY D. RUBBO AND MARJORIE E. DAVEY,[2]

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THE present investigation has proceeded along purely practical lines, with the view of indicating to the clinician and to the pharmacist how penicillin may be handled outside hospital routine. We have been concerned with the effect of temperature on the stability of penicillin "Commonwealth" in solution, and with the effect of aging of these solutions, of bacterial contamination and of admixture with acridine drugs. In addition, a new ointment base and powder base have been suggested for local application.

From the experience gained in this study we have come to the conclusion that no useful purpose is served by retaining some of the rigid precautions of storing and of dispensing which now characterize the use of penicillin. For instance, penicillin in solution is just as stable at room temperature as at 0° to 4° C. for several days, and approximately 70% to 80% of its original activity is present for at least two days after it has been dispensed. Also it has been shown that air-borne bacteria fail to grow in clinical solutions and hence fail to inactivate the drug. Accordingly chance contamination of a solution intended for local application is unlikely to cause serious deterioration in activity.

Experimental Investigation.

Penicillin "Commonwealth" as the sodium salt was used; 5-aminoacridine hydrochloride ("Monacrin") and proflavine sulphate were selected as typical acridines. Antibacterial activity was determined by the serial dilution method and in special cases by the Oxford cup assay. The medium used throughout was 10% serum broth, pH 7·3. The organisms used were *Staphylococcus aureus* and *Escherichia coli* (acridine research strains). The inoculum was 0·03 millilitre of an eighteen to twenty-four hour serum broth culture incubated at 37° C. The end-point of bacteriostasis was taken as that dilution of penicillin, expressed in units per millilitre of medium, or of acridine, expressed in concentration of base, which completely prevented the development of a visible turbidity, or a sediment which on shaking produced turbidity, after twenty-four hours' incubation.

Stability of Penicillin in Saline Solution.

It is generally accepted that the rate at which penicillin in solution loses activity depends on a number of factors, such as pH, aging, temperature, bacterial contamination, trace metals and inherent instability peculiar to different batches. Abraham and Chain (1942) state that in aqueous solution penicillin is most stable between pH 5·5 and 7·5 and retains its activity for several months at 2° C. and for several weeks at 25° C. It would appear that the optimal pH for keeping penicillin solution would be about pH 6·0 to 7·0.

Normal saline solution may vary over a wide range of pH (4·5 to 8·9) with an average reading of 8·0. It might be supposed that saline solutions at these extremes of pH would be quite unsuitable for preparing penicillin solutions. This would be so were it not for the fact that penicillin possesses a buffering capacity of its own. Of the many solutions of penicillin dissolved in different specimens of saline solution, none had a pH below 6·5 or above 7·8, notwithstanding the fact that some specimens of saline solution had a pH as low as 4·4 and others as high as 8·7. The average pH of penicillin in saline solution irrespective of strength lies between 7·4 and 7·6.

[1] Submitted for publication, April 6, 1945.
[2] Working with the aid of a grant from the National Health and Medical Research Council.

These findings suggest that a more appropriate aqueous solvent would be an isotonic saline solution buffered to pH 7·0. A formula was constructed fulfilling these conditions, and a comparison was made of the stability of penicillin in this solvent and in saline solution. The results, both by the serial dilution technique and by the Oxford cup assay, disclosed no significant advantage of the buffer solution over the saline solution. Therefore, it appears that saline solution is adequate as a clinical solvent which provides no peculiar difficulties in preparation.

The problem of the stability of penicillin in solution at various temperatures has become somewhat confused, in that the clinician has been forced to adopt the precautions of the research worker in preserving his solution. The question most pertinent to this subject is to inquire into the rate of loss of activity induced by storage at different temperatures. With this end in view a great number of bacteriostatic tests has been carried out. The conclusion has been reached that no significant difference in rate of loss of penicillin in solution can be detected over a wide temperature range from 4° C. to 37° C. during the first forty-eight hours of storage. The results of a typical experiment are shown in Table I.

TABLE I.[1]

The Stability of Penicillin "Commonwealth" at Various Temperatures.

Preparation.	Temperature of Storage.	Solvent.	pH.	Age of Preparation in Days.							
				0	1	2	3	4	5	6	7
Penicillin, 250 units, per millilitre ..	4° C.	Saline solution.	7·3	6[2]	5	5	5	5	5	5	5
	18° C.			6	5	5	5	5	5	4	4
	37° C.			6	5	5	4	4	3	3	3
Penicillin 250 units per millilitre ..	4° C.	"Monacrin", 0·1% in water.	6·5	6[2]	6	6	5	5	6	6	5
	18° C.			6	6	5	5	5	5	5	5
	37° C.			6	6	5	5	5	5	5	5

[1] Highest dilutions completely preventing visible growth of *Staphylococcus aureus* in twenty-four hours; medium, serum broth, pH 7·3.
[2] Key to penicillin dilutions.

0 signifies growth in lowest dilution, that is, 1.
1 signifies inhibition of growth in 12·5 units per millilitre.
2 signifies inhibition of growth in 6·25 units per millilitre.
3 signifies inhibition of growth in 3·2 units per millilitre.
4 signifies inhibition of growth in 1·6 units per millilitre.
5 signifies inhibition of growth in 0·8 units per millilitre.
6 signifies inhibition of growth in 0·4 units per millilitre.
7 signifies inhibition of growth in 0·2 units per millilitre.
– signifies experiment not done.

It will be seen that after an initial fall in activity, the potency of penicillin in saline solution was identical during the first two days, irrespective of the temperature at which the solution was stored. After this period the degree of inactivation gradually increased with the higher temperatures, so that after three days considerable loss had occurred at 37° C., after five days a similar loss had occurred at room temperature, but after seven days at 4° C. the activity was the same as that after one day. In 0·1% "Monacrin" solution similar results were obtained, except that the activity seemed to be better maintained than in the saline solution.

Admittedly it is difficult to estimate with accuracy by the serial dilution method the extent of loss in activity. Any manœuvre to perform more closely spaced dilutions than the doubling dilution will be defeated by the indefinite end-point in the growth of the organism. Accordingly, confirmation of these results was sought in the more accurate Oxford cup assay.

The results of the cup assay were somewhat variable, and disclosed that different batches of penicillin possessed different stabilities independent of controllable physico-chemical factors. For instance, in one experiment over 80% of the original activity was found in saline solution and in 0·1% "Monacrin" solution after seven days at 0° C. and at 24° C. A second experiment with a different batch showed 80% of the original activity present after four days at both temperatures, whereas in a third experiment 80% of the original activity was present only after one day at both temperatures.

A reasonable conclusion to be drawn from both sets of experiments is that the majority of penicillin solutions will retain 70% to 80% of their original activity for at least forty-eight hours, irrespective of whether they are kept at room or ice-box temperature.

Bacterial Contamination of Penicillin Solutions.

Great stress has been placed on the deleterious effect of air-borne contamination of penicillin solution. One reason is that injection of contaminated material may lead to local abscess formation or to bacteriæmia, and the second reason is that the infecting organisms may seriously lower the unitage of the solution. With regard to the first contingency, nothing less than bacteriological sterility is demanded of any injectable material; consequently this comment is not apposite to penicillin itself. On the other hand, the question of loss of potency in clinical solutions of penicillin by chance infection with air-borne bacteria is worthy of some discussion.

There appear to be only three possible mechanisms by which bacteria may inactivate penicillin, namely: (i) inactivation by specific enzymes (penicillinase); (ii) inactivation by non-specific enzymes (utilization of penicillin as a nutrient); (iii) inactivation by metabolic products (acids or amines). Whichever mechanism is used by an organism, some growth must take place, and it is pertinent to ask whether organisms can grow in therapeutic penicillin solutions. Some experiments have been performed to elucidate this point.

Penicillin solutions, crude and therapeutic, were inoculated with a wide variety of resistant organisms, of which three were known to produce penicillinase. The inoculum was approximately 10,000 cells per millilitre of solution and was harvested from twenty-four hour agar slopes. The tubes were incubated for three days at 37° C. and the degree of growth was assessed by turbidity. The cultures were then filtered and the penicillin content was determined by the serial dilution method. Uninoculated controls were included in all experiments.

The experiments were repeated with a much heavier inoculum, namely, 2,000,000,000 cells per millilitre.

The results are shown in Table II. It will be noted that all organisms grew freely in crude penicillin—that is, metabolism solution—but failed to produce any turbidity in the therapeutic solutions. Furthermore, the loss of penicillin in the inoculated tubes was no greater than that found in the controls, in spite of the fact that most organisms were still viable at the conclusion of the incubation period. It is worth noting that the three penicillinase-producing strains caused no greater destruction than the penicillinase-non-producing types.

On the above data it is quite evident that the ability of an organism to grow in crude penicillin is no indication of its ability to grow in therapeutic solutions. In both experiments the conditions favoured the growth of the organism to a degree which would never be realized in therapy, namely, a heavy inoculum and prolonged incubation at an optimal temperature. The significance of these findings will be discussed later.

Compatibility of Penicillin and Acridines in Solution.

The optimal treatment of local sepsis due to a mixed bacterial flora is undoubtedly a combination of penicillin with some antiseptic capable of controlling Gram-negative bacilli, such as coliform and Proteus types. Since the

TABLE II.
Artificial Contamination of Penicillin Solutions.[1]

Organism.	Solution. I. Crude Penicillin, 90 Units per Millilitre.	Solution. II. Sodium Penicillin, 90 Units per Millilitre.	Solution. III. Sodium Penicillin, 250 Units per Millilitre.	Activity of Solution III after Incubation. Sterile Control.	Activity of Solution III after Incubation. Inoculated Solution.
1. *Escherichia coli*	++[2]	−[2]	−	3[6]	4
2. *Escherichia coli*	++	−	−	3	5
3. *Escherichia coli*[4]	++	−	−	3	5
4. *Aerobacter* Type 1[4]	++	−	−	3	Not tested
5. *Aerobacter* Type 2[4]	++	−	−	3	2
6. *Aerobacter* Type 2[4]	++	−	−	3	4
7. *Aerobacter* sp.[4]	++	−	−	3	5
8. *Achromobacter* sp.	++	−	−	3	4
9. *Pseudomonas aeruginosa*[4]	++	−	−	3	5
10. *Pseudomonas aeruginosa*[4]	++	−	−	3	4
11. *Pseudomonas aeruginosa*[4]	++	−	−	3	3
12. *Proteus* sp.	++	−	−	3	5
13. *Bacillus subtilis* (N.C.T.C. No. 6346)[3]	++	−	−	3	5
14. *Bacillus* sp.[3 4]	++	−	−	3	3
15. *Bacillus* sp.[5]	++	−	−	3	2
16. *Bacillus subtilis*	++	−	−	3	4
17. P. organism[5]	++	−	−	3	2
Average	—	—	—	3	4

[1] Inocula: 10,000 and 2,000 × 10^6 organisms; incubation perod: three days at 37° C.
[2] "++" = good growth; "−" = no growth.
[3] Numbers 13, 14 and 15, penicillinase producing.
[4] Numbers 2, 3, 4, 5, 6, 7, 9, 10, 11 and 14, contaminants isolated during manufacture of penicillin.
[5] P. organism: laboratory contaminant of penicillin solution; Gram-negative motile bacillus possessing oval and subterminal spores.
[6] Penicillin dilutions.

acridine drugs are as effective as any yet discovered in the latter connexion, it was considered worth while to establish whether these two very dissimilar drugs were compatible with one another. Accordingly, 5-aminoacridine hydrochloride ("Monacrin") and proflavine sulphate solutions were mixed with penicillin and the antibacterial activity of these mixtures was compared with the separate solutions of each. The penicillin content was assessed by titration with *Staphylococcus aureus* as test organism and the acridine content with *Escherichia coli*. The results are shown in Table III.

It will be seen that the acridines do not affect the anti-staphylococcal activity of penicillin, nor does penicillin affect the action of the acridines as determined by their anticoliform action. However, it will be seen that proflavine sulphate causes serious inactivation of penicillin if its acidic pH is not neutralized by the addition of one-quarter of its weight of sodium bicarbonate (Albert and Gledhill, 1944). These results have been confirmed by the Oxford cup assay.

It may be remarked here that immediately penicillin is mixed with an acridine a turbidity develops and later a yellow precipitate forms. This precipitate is devoid of activity and may be filtered off. It probably consists of inert pigment. As the calcium salt of penicillin would precipitate in addition calcium sulphate in the presence of proflavine sulphate, "Monacrin", which is 5-aminoacridine hydrochloride, would be the more desirable acridine for combination with penicillin.

Penicillin in Powder and Cream.

Various excipients have been used to dilute penicillin for local application, but none can be considered entirely satisfactory. The sulphonamides are expensive, and in the presence of pus do not contribute to the therapeutic value of the preparation. Furthermore, they may give rise to local and general toxic phenomena, so that their use should be restricted to cases in which their employment is indicated on therapeutic grounds. Dried human serum has been successfully used at a military hospital, but this product is too expensive and too scarce to be generally useful. Glucose may serve as a useful diluent; but it may also stimulate growth of organisms present in the lesion. The possibiliy of using starch as an excipient has been

TABLE III.
Compatibility of Penicillin with Acridine Drugs.[1]

Content of Solution.	pH.	Storage Temperature.	*Staphylococcus aureus*: Penicillin Assay. Age of Solution (Days.) 0	1	4	8	*Escherichia coli*: Acridine Assay. Age of Solution (Days). 0	1	4
Penicillin, 250 units per millilitre	7·2	18° C.	6[2]	5	5	5	0[3]	0	0
"Monacrin", 0·1%	6·0	18° C.	2a	2a	2a	2a	2a	2a	2a
Proflavine, 0·1%	2·0	18° C.	2a	2a	2a	2a	1a	—	—
Penicillin, 250 units per millilitre; "Monacrin", 0·1%	6·5	18° C.	6	6	6	5	2a	2a	2a
Penicilln, 250 units per millilitre; proflavine, 0·1%	2·8	18° C.	5	2	2	2	1a	—	—
Penicillin, 250 units per millilitre; proflavine, 0·1%; sodium bicarbonate, 0·025%	5·6	18° C.	6	5	5	5	1a	—	—

[1] Highest dilutions completely preventing visible growth in twenty-four hours; medum: serum broth, pH 7·3.
[2] Penicillin dilutions.
[3] Acridine dilutions: "1a" signifies inhibition of growth in one in 20,000 dilution; "2a" signifies inhibition of growth in one in 40,000 dilution; "−" signifies no experiment made.

explored experimentally in this study. Starch, sterilized by dry heat, possesses most of the attributes desired of an excipient, as recently set out by Officer, Loewenthal and Perry (1944).

Preliminary to the bacteriological experiments on starch, some physical and chemical properties have been superficially examined.

Sterilization can be effectively achieved by dry heat at 150° C. for thirty minutes when spread in shallow layers.

The reaction is neutral; the pH of serum is unaltered by the addition of starch.

No reducing sugars are present after sterilization; there is no evidence of dextrin formation.

Starch is practically insoluble at 37° C. in serum. This may constitute a disadvantage; but absorption would take place by phagocytosis and hydrolysis.

There is no evidence of gel formation at 37° C.

Loss of weight on dry sterilization is approximately 13% to 15%, whilst gain in weight when exposed to air is about 7% after seven days and on standing for seventeen days up to 200%. A penicillin (sodium salt) and starch mixture showed identical gain in weight with that of sterile starch.

The following powders were dispensed under aseptic conditions, in pestle and mortar, and stored at room temperature (16° to 18° C.) in screw-capped jars for a period of seventeen days; the powders were composed as follows: (i) number I: penicillin, 500 units per gramme of starch; (ii) number II: powder I with "Monacrin" (0·1%); (iii) number III: powder I with "Monacrin" (0·5%); (iv) number IV: powder I with "Monacrin" (1·0%).

Assays were performed by the serial dilution technique and from the data for powders I and IV (Table IV) it is apparent that penicillin is stable in starch and in starch-"Monacrin" powders. The presence of the acridine greatly enhances the activity against the Gram-negative types of bacteria.

TABLE IV.

The Stability of Penicillin and "Monacrin" in Starch Powder: Weight of Powder per Millilitre of Serum Broth Required to Inhibit the Growth of the Test Organisms.

Powder. (See text.)	Time of Storage at 18° C. (Days).	*Staphylococcus aureus* (Penicillin Assay). (Milligrammes.)		*Escherichia coli* (Acridine Assay). (Milligrammes.)	
		Expected.	Found.	Expected.	Found.
Powder I	0	1·6	1·6	>100	>100
	17	1·6	1·6	>100	>100
Powder IV	0	1·6	1·6	10	10
	17	1·6	1·6	10	10

By frosting the surface of heavily inoculated agar plates with powder IV (1% of "Monacrin") it was possible to inhibit the growth of Proteus and coliform bacilli, but not of *Pseudomonas aeruginosa*. On these results it appears that a starch excipient for penicillin may be worthy of a clinical trial, and particularly because the cost is only a fraction of that of the cheapest sulphonamide.

Few ointment bases are available in Australia in which the activity of penicillin is well maintained. "Barrier Cream" (Faulding) has been found to be satisfactory. A preliminary investigation of neutral "Tegin", commercially described as glyceryl monostearate, suggests that it would be quite a suitable vehicle for penicillin. "Tegin", in a 10% to 12% concentration in water, forms a smooth white cream which is greaseless and readily penetrates the skin. It withstands autoclaving without change in character. The pH of several batches in a concentration of 10% in water varied between 7·0 and 7·4. Oils of aqueous solutions can be incorporated into this cream without difficulty. The results of the following assays (Table V) show that penicillin is as stable in "Tegin" as it is in "Barrier Cream".

No significant difference in penicillin activity was demonstrated in either cream on storage at room and ice-box temperature. It may be concluded that the two creams are practically identical so far as the keeping qualities of penicillin are concerned.

TABLE V.

The Stability of Penicillin in Creams: Weight of Cream per Millilitre of Serum Broth Required to Inhibit the Growth of Staphylococcus aureus *in Milligrammes.*

Cream.	pH.	Days of Storage at 4° C. and 18° C.			
		0	2	5	18
"Tegin", containing 500 units per gramme ..	7·3	0·8	0·8	0·8	1·2
"Barrier Cream," containing 500 units per gramme	7·1	0·8	0·8	1·2	1·2

Discussion.

The points to be raised here are few. It has been shown that penicillin "Commonwealth" is far more stable in aqeuous solution than is generally recognized. We consider that the clinician may confidently use penicillin solutions stored at room temperature for at least two or three days. The activity at the end of the second day would probably be 70% to 80% of the original activity, so that the dose could be adjusted accordingly. In our experience this would be a conservative estimate of the stability in solution; but in view of the variability of different batches of penicillin, a safe mean value has to be selected. It is worth noting that Kirby (1943) arrived at similar conclusions in respect to American types of penicillin. We believe that these results show the desirability of marking each batch of penicillin with a figure indicating its stability in saline solution at average room temperature. In this way a great deal of uncertainty would be removed, the inconvenience of storage at low temperatures would be unnecessary and the discomfort of the patient less. It is well known that the injection of chilled solutions is far more painful that that of warm ones.

Penicillin intended for local application, either therapeuticaly or prophylactically, may be dispensed in a variety of ways. In aqueous solution it may be combined with acridines, preferably 5-aminoacridine ("Monacrin") and stored at room temperature for five to seven days. The strength should be 500 units per millilitre to allow for deterioration over this time. In creams penicillin appears to be "clinically" stable for at least five to seven days at room temperature, and in powder (starch) for an even longer period of time.

In respect to the aseptic precautions to be taken in dispensing solutions for local application—namely, treatment of infections of eyes, ears, skin and mucous membranes—we consider that bacteriological sterility would be advisable and that the patient should be instructed to maintain this sterility by hot-water sterilization of droppers and by the minimal exposure of the solution to the air. If these simple precautions are adopted the clinician may be assured that unitage will not be lost owing to slight chance contamination, since it has been shown that bacteria will not grow in clinical solutions.

In conclusion we advócate that penicillin should be used in a manner consistent with its properties, and in our experience these properties are such as to warrant some revision in the present recommendations.

Summary.

1. Penicillin "Commonwealth" in saline solution retains 70% to 80% of its activity at room temperature for at least two days.

2. Penicillin is compatible with acridine drugs, although proflavine sulphate must be neutralized.

3. Penicillin will not support the growth of a wide variety of penicillin-fast bacteria. Hence clinical solutions are not inactivated by them.

4. Penicillin is stable in a cream base, neutral commercial glyceryl monostearate, and in starch.

5. A stability test in saline solution at room temperature is recommended for each batch of penicillin.

Acknowledgements.

We wish to express our thanks to the National Health and Medical Research Council for making supplies of penicillin available, to Dr. F. G. Morgan and members of the Penicillin Assay Section of the Commonwealth Serum Laboratories for performing cup assays quoted herein, and to Mr. G. Anderson, also of the Commonwealth Serum Laboratories, for cultures of certain penicillin-fast bacteria.

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ON THE PRICKLY HEAT.

By FRANKLIN R. FAY,
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AND

ERIC SUSMAN,
Royal Australian Naval Reserve, Sydney.

Falstaff: Think of that, a man of my kidney, think of that, that am as subject to heat as butter.

Shakespeare—"The Merry Wives of Windsor."

PRICKLY HEAT, or *miliaria rubra* or *lichen tropicus*, is an annoyance rather than a disease. Its ætiological stage is always set in the same pattern. It appears under hot, humid, environmental conditions, and invariably and completely disappears in cool weather. Its general clinical features are clear cut, and the diagnosis is easy. The distribution of the rash varies from patient to patient, although it nearly always affects the shoulders and the upper part of the arms; but it never deviates from its dimorphic character. This is a combination of small red papules and tiny vesicles. Sometimes the one, sometimes the other dominates the picture. The vesicles look and feel like sago grains in miniature. The malady is monosymptomatic. Patients complain of pricking, tingling sensations in the affected area. Itching, invariably stressed in dermatological text-books, is unusual, the eruption being vexatious rather than irritating. It is certainly not itchy in the sense that scabies is itchy. In our experience, superadded infection and scratch dermatitis never occur. In an attempt to assuage the bizarre sensations, the sufferer tends to rub or stroke the affected area with the palm of the hand, in much the same way as an area of paræsthesia, from whatever cause, is stroked, fondled or caressed. The finger nails are not used. In a supersensitive patient sleeplessness may result, but in the great majority of cases there are no constitutional symptoms, nor is there any loss of bodily or mental vigour arising from the rash as such. There are no complications, and relapse, given the appropriate surroundings, is the rule.

Allen and O'Brien[1] have recently described a syndrome which they call "tropical anidrotic asthenia". It is apparently a sequel of, or "hangover" from, an attack of *miliaria rubra* that has occurred about four weeks previously. From a careful reading of the article, the pathological basis of the syndrome seems to be a clogging of the sweat ducts by epithelial debris, due to a previous attack of prickly heat. The treatment of this specific asthenia is an extremely pleasant one—six to twelve weeks in a cool, ventilated ward. Rather than this sybaritic rest cure, rational therapy would seem to consist of the vigorous and manly use of a hard nail brush, with plenty of soap and water, thus achieving a return to full duty within the hour. The preliminary report of this syndrome is not convincing, and we look forward keenly to its amplification. The syndrome does not seem to occur in naval medical practice. As stated already, after more than a nodding acquaintance with this mildest of afflictions we are quite ignorant of any sequel to it. It is to be hoped that the sailors do not get to hear of this syndrome or its treatment. It might become fashionable in naval circles, and the Senior Service can scarcely afford the luxury diagnosis "asthenia". In His Majesty's ships serving in the tropics, prickly heat in susceptible subjects has come to be regarded as an inevitable minor ill, worthy only of routine palliative measures for its alleviation. Under ordinary cruising conditions one is not able to predict an outburst of the trouble in regard to its frequency, its relative severity or the selectivity of personnel.

CLINICAL SURVEY.

It was the capricious behaviour of this malady that decided us to make the following investigations in an attempt to discover the factors (if any) of susceptibility in a small group of our ship's company. Advantage was taken of a recent tedious and prolonged naval action, extending over six days and nights, to make some clinical observations on 46 naval officers, living and working under the standard conditions summarized later. Their environmental conditions of work and living prior to the period of action were studied also, with a view to the obtaining of data on factors which varied considerably from individual to individual. These are mentioned later. The standard living and working conditions, referred to earlier, were as follows:

1. *State of Ship.*—The ship was "closed up"; that is to say, it was divided into compartments by water-tight doors, and all upper deck hatches were closed.

2. *Dress.*—All subjects wore long-sleeved shirts, long trousers, boots and socks (cyclist fashion) and, when necessary, "anti-flash" gear. Anti-flash gear consists of long gauntlet cotton gloves and a covering for the head and neck—a cowl, shaped like a Balaclava cap (Figure I).

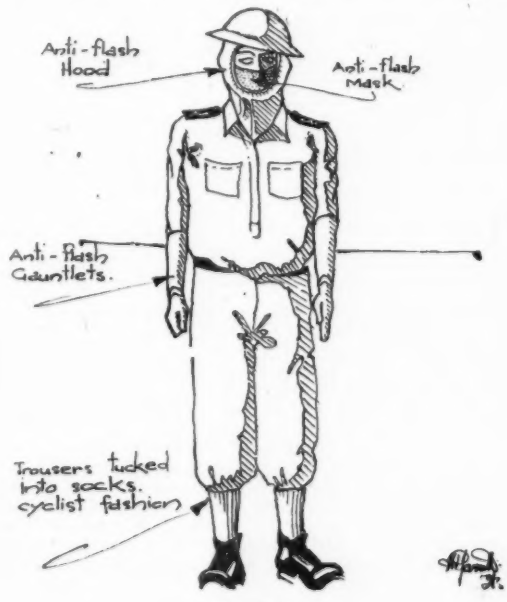

FIGURE I.

3. *Temperature of Sea Water.*—The temperature of the sea water during the six days' observation period averaged 87° F.

4. *Conditions Inside the Ship.*—The average temperatures inside the ship were as follows: dry bulb, 94° F.; wet bulb, 81° F. The humidity was 57%.

5. *Position of the Ship.*—The ship was within 4° of the equator.

6. *Diet.*—A standard mixed diet was taken with no obvious vitamin lack. There was practically no consumption of alcohol during the period under review.

Analysis of the Cases.

Prior to the action period, of the 46 officers, 18 had prickly heat. These men lived under similar conditions of diet and cleanliness and good health. During the six-day action period, eight more developed the rash, and the significance of this increase is discussed below. Eighteen variable factors were studied in subjects with and without *miliaria rubra*.

It is realized from the start that the numbers concerned are small, and therefore subject to the usual statistical fallacies. But they do give some basis for general conclusions. The study of large numbers would have involved too great a disturbance of the ship's war-time routine. The factors studied are listed below, together with the tables of our results and our conclusions drawn from them.

Age.

Of the 46 subjects, 23 were aged under thirty years and 23 were aged over thirty years. Of the 23 aged under thirty years, six had a rash; of the 23 aged over thirty years, 12 had a rash. These figures indicate that subjects aged over thirty years may be more prone to the condition than younger subjects. Lessening in the efficiency of the heat-regulating mechanism, coincident with advancing years, is the speculative explanation of these findings.

Weight.

Of the 46 subjects, 37 weighed from 9 to 13 stone, and 9 weighed more than 13 stone. Of the 37 weighing below 13 stone, 15 had a rash; of the 9 weighing more than 13 stone, 3 had a rash. Overweight appears to have no bearing on the incidence of prickly heat.

Blood Group.

Of those suffering from the rash, the majority belonged to the group O (IV). This percentage was much higher than the percentage distribution of this blood group among all officers being studied.

TABLE I.
Blood Group.

Blood Group.	Rash.	No Rash.	Total.
AB (I)	0	1	1
A (II)	7	14	21
B (III)	0	3	3
O (IV)	11	10	21

Of the 46 officers, 21 belonged to blood group O (IV) and 21 to group A (II). Of those without the rash, the majority belonged to blood group A (II). This suggests, perhaps, that persons belonging to blood group A (II) are less liable to prickly heat than those of other blood groups. Groups AB and B have been disregarded.

Complexion.

Of the 46 subjects, 13 were of light complexion, 15 of medium complexion and 18 of dark complexion. Of those with light complexions, seven had a rash; of those with medium complexions, four had a rash; of those with dark complexions, seven had a rash. It seems that fair-skinned persons are more prone to prickly heat than dark-skinned persons.

Exposure to the Sun.

On exposure to the sun, 33 of the subjects became tanned and 13 became burnt. Of the 33 who became tanned, 13 had a rash; of the 13 who became burnt, 5 had a rash. It seems that whether or not a person is burnt or tanned by the sun has no bearing on the incidence of prickly heat.

Absence of Sun-Tan Prior to Tropical Environment.

Twenty-one of the subjects were sun-tanned prior to entering the tropics and 25 were not. Of the 21 sun-tanned subjects, 6 had a rash; of the 25 who were not sun-tanned, 12 had a rash. A sun-tanned (pigmented) skin seems to be more resistant to *miliaria rubra* than an unpigmented skin. A few subjects have the rash on the buttocks—an area rarely sun-tanned.

Psychological Factor.

From the psychological point of view, six of the subjects were classed as "phlegmatic", 19 were classed as "normal", and 21 were classed as "highly strung". Of the "phlegmatic" subjects, two had a rash; of the "normal" subjects, seven had a rash; of the "highly strung" subjects, nine had a rash. As one studies the incidence of the rash in the different psychological groups, a definite increase is observed over the range "phlegmatic"-"normal"-"highly strung". Although no great variation occurs, the indications are that *miliaria rubra* is more apt to occur in those subjects who are highly strung. Difficulties of classification, however, make one wary in drawing any conclusions.

In parenthesis, it may be stated that psychological classification is by no means an easy, clear-cut matter. Our sorting of these 46 men is of some interest. We separately made our classification and were agreed about the 6 phlegmatic and the 21 highly strung subjects. Our separate classification of the 19 "normals", however, by no means coincided, and final classification was arrived at only after consultation and discussion.

Sweat Reaction.

As the sweat of 40 subjects had an acidic reaction and that of only 6 had other types of reaction, it is hazardous to draw any conclusion from these figures. Our facilities for examining the pH of sweat were of the crudest—litmus paper.

TABLE II.
Sweat Reaction.

Sweat Reaction.	Rash.	No Rash.	Total.
Acid	16	24	40
Neutral	1	1	2
Alkaline	1	3	4

Salt Intake.

Salt on Food.—The indications are that a small to moderate salt intake helps to prevent the rash, but a large salt intake favours its appearance.

TABLE IIIA.
Salt on Food.

Salt Intake.	Rash.	No Rash.	Total.
None	1	3	4
Little	3	7	10
Moderate	3	11	14
Much	11	7	18

Salt Tablets.—Salt tablets weigh 14 grammes each and were available at all times. In the table the number of men taking over four per day is too small to be of use statistically. The indications are similar to those learnt from Table XA—that is, a low or moderate intake of up

TABLE IIIB.
Salt Tablets (14 Grammes Each).

Tablets per Day.	Rash.	No Rash.	Total.
None	9	18	27
0·5 to 1	2	5	7
2 to 3	6	4	10
4 to 5	1	0	1
6 and over	0	1	1

to to 14 grammes of salt per day helps to avoid the "disease", whereas an intake of over 28 grammes per day aggravates the condition.

It is suggested that this aggravation of the *miliaria rubra* by a high salt intake is due to the subject's over-compensating for salt lost by sweating, with the result that there is an upset in the osmotic balance in the blood followed by an increased rate of sweating.

Underclothing.

Fifteen subjects wore no underclothing, 30 wore cotton underclothing and one wore silk underclothing. Five of the first group had a rash and 13 of the second group; the man who wore silk underclothes had no rash. As nearly everyone wore either no underclothes or cotton underclothes, the material could not be investigated. However, among those wearing no underclothes there was a lower incidence of the condition, probably due to better ventilation of the body and to absence of moist, clinging singlets and "shorts".

Soap.

It was impossible to draw any deductions from observations made on the types of soap used. We had no facilities for analysing the alkaline content of soap.

Clothing.

"Tropical rig" consisted of "shorts", shirt and sandals. The wearing of the shirt was optional, so this was studied. The observations were made on clothing worn on duty, but not in action. Of the 31 men who wore shirts, 12 had a rash; of the 15 who wore no shirt, 6 had a rash. The incidence of the rash appeared to be unaffected by the wearing of shirts.

Observations made on clothing worn off duty showed that of 9 men who wore shirts, 3 had a rash, and of 37 men who wore no shirt 15 had a rash. It was noticed that during the six days' action period, eight men developed the rash who had previously been free from it, and five men complained of aggravation of an existing rash. This was most probably due to being constantly in battle dress, which does not allow nearly so much ventilation as does an open-necked shirt and short pants.

Occupation.

The number of officers doing manual work (two) was so low that the comparison with those doing administrative work (44) was valueless. Most sufferers, however, complained of an aggravation of symptoms on exercise, most probably due to increased dilatation of the surface capillaries with increase in sweat production and in skin temperature at the humidity obtaining under equatorial conditions.

Working Hours.

Thirty-four men worked up to eight hours a day, and nine of these had a rash; eleven worked from eight to twelve hours a day and eight had a rash; one man worked for more than twelve hours a day and he had a rash. It is observed that, as the number of working hours increases, so does the incidence of *miliaria rubra*. Most men spend their leisure hours on deck in the shade with their shirts off. The number of working hours was decidedly raised during the action period, and this may be a factor in the increase in the number of sufferers at that time.

Place of Work.

Fifteen men worked on the upper deck and four had a rash; 24 worked between decks and 12 had a rash; seven worked in the engine or boiler room and two had a rash. The incidence of prickly heat was thus higher among those working between decks, where the temperature was 90° to 100° F. and the humidity about 60%. The low incidence of the disorder in the engine and boiler rooms may be due to the fact that the humidity there is only 45%, though the temperatures varies from 110° to 120° F.

Average Temperatures of Place of Work.

Except for the highest temperatures when the humidity is low (*vide supra*), the incidence of the condition is increased when the temperature of the place of work is raised. In temperatures below 90° F., the majority are free of the rash.

TABLE IV.
Temperatures.

Temperature. (Degrees F.)	Rash.	No Rash.	Total.
80 to 90	10	17	27
90 to 100	5	5	10
100 to 110	0	2	2
110 to 120	3	4	7

Average Humidity of Place of Work.

The highest humidity is on deck, where it is 60% to 70%, but there the temperature is lowest—80° to 90° F. These results are somewhat complex to interpret because of their relation to temperature, and because of the small numbers of cases studied. It is suggested that as the humidity rises to 60%, the incidence of the rash increases also, but above this (for example, on deck) the incidence falls considerably. These results are most inconclusive.

TABLE V.
Humidity.

Humidity.	Rash.	No Rash.	Total.
40% to 50%	3	4	7
50% to 60%	13	17	30
60% to 70%	2	7	9

Sleeping Place.

Twenty of the men slept on deck and seven had a rash; 26 slept below deck and 11 had a rash. Many people sleep on deck on stretchers, which is much cooler than sleeping below, as the ship retains much of the heat from the sun, engines *et cetera*. The observations indicate that the incidence of the rash is slightly higher among those sleeping below decks.

General Observations.

During the action period five officers who already had the rash complained that its severity increased. Eight officers who had not had the rash previously now developed it, thus bringing the total number of officers afflicted to 26. An analysis was made of the exciting factors to the disorder of these officers who developed a rash. Only the factors which we found significant were studied. The results were not very helpful, except that the wearing of battle dress, and longer working hours, appeared to act as the "last straw".

Although no comparative figures were obtained, observation showed that the "lower deck" men were less affected by prickly heat than were the officers—in fact, the difference was most noticeable. As the greatest proportion of the crew work on deck and a large proportion in the engine and boiler rooms, only a small number are between decks in offices, and it was noticed that it was this last group of men who had the affliction at its worst. It has already been shown that the lowest incidence is found on deck and in the boiler rooms, so a large factor in the difference in incidence between officers and men is the site of work. Other factors coming into the question are: (i) ratings on the average wear less clothes than officers; (ii) officers' quarters aft are the worst ventilated parts of the ship.

The areas of skin most commonly affected seem to be those areas where the moist, sweaty clothing sticks—that is, the shoulders, the upper part of the arms, the back, under watch straps and around the waist and at the top of the trousers. Sweat stagnation and accumulation (absence or lack of evaporation) appear to play a not inconsiderable part in determining the site of the lesions.

Whether or not there is an allergic factor has to be considered, as it is suggested by the case of Lieutenant-Commander M., who is an engineer, aged thirty-five years,

with a fair skin. His rash is pronounced during his period in the engine-room; it clears considerably almost as soon as he comes out on deck, and completely disappears after he has slept on deck. He may be allergic to fuel oil.

Conclusion.

From the investigations carried out, we have found that the factors examined may be classified as follows:

1. Factors which increase the liability of a person to prickly heat are: (*a*) age over thirty years, (*b*) fair skin, (*c*) "highly strung" type of mental make-up, (*d*) large salt intake, (*e*) working and sleeping below decks, (*f*) high temperatures of place of work, (*g*) long working hours, (*h*) battle dress or poorly ventilated clothing, and (*i*) perhaps the fact of belonging to blood group O (IV).

2. Factors which reduce the liability of a person to prickly heat are: (*a*) age below thirty years, (*b*) previous sun tan, (*c*) loose clothing, (*d*) open-air working and sleeping, (*e*) low temperatures of place of work, and (*f*) perhaps the fact of belonging to blood group A (II).

3. Factors having no effect on the incidence of prickly heat are: (*a*) weight, (*b*) sweat reaction, (*c*) wearing of open-necked shirts, and (*d*) the effects of the sun on the subject's skin.

4. Factors which from our investigations gave inconclusive results were: (*a*) type of work, (*b*) humidity, (*c*) brand of soap used.

These various factors do not seem to be a cause in themselves, but when present they affect a susceptible person as indicated. The disease seems to be more constitutional than to be due to any one factor in particular.

TREATMENT.

Prophylactic Notions.

Almost every non-sufferer expressed a positive *ex cathedra* opinion about his freedom from prickly heat, usually stated with some degree of self-satisfaction. These various opinions were, of course, mutually contradictory. They may be compared with the two nonagenarians when interviewed by the Press: the one puts down his longevity to abstemious sexual, alcoholic and smoking habits; the other, a self-confessed libertine, maintains that port and cigars have kept him alive for the last thirty years. The same conflicting and contradictory opinions were obtained about *miliaria rubra*. One subject swears by two or more salt-water baths per day; the other says his escape is due to the absolute avoidance of sea-water. One gives credit to his thinness, another to his corpulence. Dietetic fads and fancies, especially by regular subscribers to literary digests, have their devotees. A particular brand of soap has its friends and enemies, and so on. There is, in point of fact, no absolute prophylaxis.

Palliative Treatment.

So far as we know, there is no cure for *miliaria rubra* except to get out of the tropics. Palliative measures are used, such as *Lotio Calamina* or a lotion containing 50% *Spiritus Vini Methylatus* with menthol and perchloride of mercury (R.A.F. lotion). Any cooling lotion will do. The individual response to these lotions is variable; some people obtain relief from only one, others from neither, others from both. We exhibited calcium lactate (60 grains three times a day) in a tolerably severe case. There was no improvement.

DISCUSSION.

The results of this inquiry have been largely disappointing. This was to be expected. The factors controlling the selectivity in any given series of cases of this trivial and inconsequential complaint must be put down to some obscure defect or abnormality in the *milieu interne* —"a man of my kidney", as the poet puts it. At the present time it is mischievous, we think, to exalt the complaint or its supposed sequelæ to the status of a "doctor-made" syndrome. Except for the exposure of some text-book[2] inaccuracies and banalities, this investigation has not achieved much. And in consideration of the light view we take towards this malady, it may be reasonably maintained that we have already made a mountain out of a mole-hill.

SUMMARY.

We have given a description of prickly heat and the relevant results of a clinical study on 46 officers serving in one of His Majesty's Australian ships, under a war-time régime, in tropical waters. Eighteen simple variable factors—anthropological, environmental and physical—are studied under more or less standard experimental conditions, and an attempt is made to postulate their effects on individual susceptibility. In the matter of treatment, a healthy stoicism towards a minor ill is, in our opinion, the correct attitude to adopt.

ACKNOWLEDGEMENT.

These remarks are published by permission of the Director of Naval Medical Services, Surgeon Captain W. J. Carr, C.B.E., R.A.N.

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PHYSIOTHERAPY IN GENERAL PRACTICE.[1]

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PHYSICAL MEDICINE includes the employment of the physical and other effective properties of light, heat, cold, water, electricity, massage, manipulation, exercise and mechanical devices for physical and occupational therapy in the diagnosis and treatment of disease. I shall not attempt to give any technical details of the frequencies and wave-lengths of the diathermy machines in use, as most practitioners have not the time or room to give short-wave treatments, more particularly as wartime regulations prohibit the use of any high-frequency apparatus outside a properly screened room.

In general, the more recently developed short-wave technique with its higher frequencies has superseded the long-wave lower frequency methods, with the exception of pelvic and prostatic applications, in which it is still the method of choice. In the treatment of the pelvic organs in women and the prostate in man, special electrodes are used in which a thermometer is incorporated enabling the local heating to be accurately measured. As a safe working rule, the more acute the condition, the lower the intensity and the shorter the treatment, which can be repeated twice or three times a day. Chronic conditions stand higher intensity and longer intervals. If the conventional long-wave diathermy is being used for other than pelvic applications, longer time for each treatment should be given than is generally in use. Hemmingway and McClendon have worked out the high-frequency resistance of human tissues in ohms, as follows:

Skin	289 ohms	Kidney	126 ohms
Fat	2,180 ohms	Liver	298 ohms
Bone	1,800 ohms	Heart	132 ohms
Muscle	110 ohms	Spleen	256 ohms

As it takes at least fifteen to twenty minutes to overcome the resistance of the skin and fat, to heat deeper structures forty to forty-five minutes are necessary.

There are three main contraindications for the use of short-wave diathermy: (i) the presence of acute inflammatory processes accompanied by fever and suppuration without drainage; (ii) the occurrence of recent hæmorrhage, such as bleeding from a gastric or duodenal ulcer, from the bowel, or the near onset or presence of menstruation; short-wave diathermy should never be given to the chest in pulmonary tuberculosis, whether hæmoptysis has occurred or not; (iii) the presence of a malignant

[1] A post-graduate lecture, delivered at the Royal Prince Alfred Hospital, Sydney, on May 22, 1945.

tumour in the area to be treated or any suspicion that a malignant tumour may be present. Special precautions should be taken in treating men who have been on active service, as the presence of unsuspected pieces of metal in the path of the current will cause acute pain. Should pain be increased when this type of patient is being treated, the machine should be turned off at once and an X-ray picture taken to see whether metal fragments are present. Infra-red and ultra-violet rays, not being in the high-frequency category, can be used anywhere outside a screened room.

There are two main types of infra-red lamps—the luminous and the black body burner. The luminous type is a tungsten gas-filled incandescent globe of either 500 or 1,000 watts. The black body type is either an iron core or coiled wire, which may be exposed or encased in fire clay. I am frequently asked by medical men which type of lamp is the more useful. It is stated that the difference between the output of the high temperature (luminous) and the low temperature (non-luminous or black body) generator is the preponderance of the short, more penetrating infra-red rays in the first group. For hospital use, when the lamps have to be wheeled to different wards, the jolting tends to break the filaments in the luminous type. This does not occur in the black body generator. Treatment with either type should be at comfortable toleration to the patient. If any stinging in the skin occurs, the lamp is too close. Great care should be taken in giving infra-red irradiation over scar tissue or where there is any impairment of sensation, as in these circumstances blisters may easily occur. The duration of the treatment should be forty to sixty minutes, and the treatment can be repeated as often as necessary. Infra-red irradiation produces an intense local hyperæmia, relieves muscle spasm and promotes absorption of local inflammatory products. As the wave-length is not penetrating to any appreciable extent, its use is limited to superficial joints and areas not covered by much muscle or fat. If it is required to heat more than three or four centimetres below the skin, short-wave diathermy must be used.

The wave-lengths of ultra-violet rays are produced by passing a high-frequency current across a mercury pool. The mercury becomes incandescent and gives the required wave-length. The action of the ultra-violet rays on the body is a chemical one. The ergosterol in the skin is activated, with greater production of vitamin *D* and consequent greater absorption of calcium.

Three factors have to be taken into consideration when one is deciding what dose of ultra-violet irradiation to give. (i) The age and type of burner being used. All burners gradually deteriorate with age, and longer exposures are required as the burners grow older. (ii) The type of skin reaction required. For tonic doses only a faint erythema should be produced, which fades in a few hours. For certain skin conditions a second or third degree of erythema is required. (iii) The type of patient being treated, whether blonde or brunette. The brunette will stand larger doses than the fair-skinned type. With the average lamp, for a tonic dose, three minutes at a distance of thirty inches are sufficient. Do not repeat the dose until all skin reaction has subsided; this is usually in three or four days.

The Uses of Ultra-Violet Light.

In England increasing amounts of ultra-violet light are being used, particularly for children. At Bermondsley before the war a solarium was established for children, and these solaria are being widely extended. Many war factories and coal mines have established ultra-violet light clinics, where shift workers who are not able to get much sunlight receive regular doses. The navy recently established a special ultra-violet depot for submarine crews, where all men in their rest periods in port are given regular doses. Children in particular respond well to ultra-violet irradiation. Care should be taken that children do not interfere with the protective goggles over their eyes when they are under the lamp. Even a short exposure of their eyes to the rays can do serious harm. The following conditions repay the application of this form of treatment.

1. Rickets and osteomalacia. The pathology of these conditions naturally suggests one of the widest fields for the use of ultra-violet irradiation.

2. Post-pertussis and post-measles cough. Many children who develop pertussis and measles in the late summer and autumn will continue to cough through the winter; wet and cold weather seems to intensify the paroxysms. Three of four exposures to ultra-violet rays will as a rule completely clear the trouble; appetite increases, and sleep is unbroken at night.

3. Generally poor physical condition. A type of child commonly met with is the thin, undernourished, restless, peevish type, with poor appetite, who is a bad sleeper—often an only child, suffering from over-indulgent parents. These children settle down quickly; they eat better, sleep better and become much more tractable.

4. Tuberculous adenitis. General tonic doses with a more intense local irradiation of the infected glands help to raise the resistance of these children.

5. Pink disease. These children recover more quickly if ultra-violet irradiation is added to their regular treatment.

6. Juvenile asthma. Many of these sufferers have an associated rhinitis or low-grade sinusitis. The routine method here consists of short-wave irradiation to the sinuses, the application of general ultra-violet irradiation, and special asthma breathing exercises. This regimen, if carried on for some time, will as a rule greatly benefit these patients. In the asthma breathing exercises special attention is paid to expiration and relaxation. Several parents have told me that they can control the child's attack if they start the breathing exercises as soon as it threatens.

Skin Diseases.

In the treatment of skin diseases by ultra-violet rays, the use of the Kromayer lamp is the method of choice. The Kromayer lamp has the burner enclosed in a circulating water bath. A glass window in the casing allows the exit of the ultra-violet rays, and the circulating water keeps the temperature of the burner sufficiently low to allow direct contact with the skin. When treatment is commenced, contact is maintained for only three seconds. The following are some of the skin diseases treated with the Kromayer lamp.

1. Psoriasis. A dose producing a second or third degree of erythema should be given to all spots on the first occasion. If the preliminary dose is too small, the lesions seem to become photoresistant.

2. *Alopecia areata.* The same conditions apply in this condition as in psoriasis. A fairly severe preliminary dose is necessary, as the areas quickly become pigmented, and pigmentation is Nature's protection against ultra-violet rays.

3. Boils and carbuncles. Preliminary irradiation by infra-red rays at low intensity, followed by a mild erythema dose of ultra-violet rays, helps to relieve pain and hastens the separation of sloughs.

4. *Herpes zoster.* Irradiation of the whole nerve involved quickly relieves the pain. Similarly post-herpetic neuralgia can be relieved by a preliminary application of short-wave therapy to the nerve root, followed by ultra-violet irradiation of the affected area. Care must be taken with the doses of short-wave therapy, as these patients often stand heat badly.

5. *Acne vulgaris.* If the rash is confined to the face only, direct application by the Kromayer lamp is best. If the rash is widespread over the back and chest, general ultra-violet irradiation is the method of choice.

6. *Lupus vulgaris.* Ultra-violet irradiation is recommended; but as this condition is seldom met with in Australia, I have not had any experience of the result.

Sinusitis.

The wider use of short-wave therapy and ultra-violet irradiation by means of the cold quartz rod has helped to bring relief to the multitude of sufferers from this most distressing and debilitating condition. Before treatment is begun, it is necessary to have adequate drainage. Surgical treatment is necessary to evacuate pus under pressure, and

to remove a deflected septum or infected turbinates. If drainage is established, very few patients cannot be relieved, and the majority are freed from their symptoms. Correct placing of the electrode is essential to obtain maximum heating into the sinus involved. The cold quartz referred to is a fused quartz rod attached to the window of a Kromayer lamp. Quartz has the property of conveying ultra-violet rays from the tip without heat and without distortion from the side of the rod. An increasing number of children are now found to be suffering from sinusitis with tracheitis, bronchitis, bronchiectasis or asthma. These children respond extremely well to a combination of short-wave therapy to the sinuses, general and local ultra-violet therapy, and asthma breathing exercises.

Aural Furunculosis.

Aural furunculosis requires low intensity treatment in the early stages, either by short-wave therapy, or if infra-red irradiation is used, with a localizer to concentrate the heat on the ear. As soon as the swelling has subsided sufficiently, the whole canal should be irradiated with ultra-violet rays from the cold quartz rod.

Otitis Media.

In many cases of *otitis media*, if the patient is examined and treated in the early stages of inflammation of the drum before bulging is present, the condition can be absorbed. If paracentesis is necessary, subsequent irradiation with infra-red rays will reduce the resultant drainage by half. Whilst dealing with conditions of the head, I must mention the great value of short-wave therapy in deep-seated eye inflammation. In iritis pain is greatly reduced or completely relieved, and many early lesions are cleared up in a few days. Treatment should be at low intensity for about fifteen minutes and repeated two or three times a day.

Fibrositis.

Fibrositis has been defined as the reaction of the fibrous tissues of the body to extraneous poisons (either bacterial or toxic) in persons constitutionally disposed to such reactions. There is a serous exudate into the affected parts followed by cell proliferation. This proliferation in the early stages takes the form of a fine network distributed throughout the area. Later these fibrils either tend to become parallel and to form bands or adhesions, or collect into nodules. These nodules are known as myalgic or trigger spots. There is always an associated muscle spasm. Some authorities state that the whole condition is muscle spasm only; but the fact remains that these myalgic spots and adhesions do develop. There are certain areas in which these spots tend to occur more frequently. These situations are: along the outer edge of the upper belly of the trapezius from the occiput to the acromion process, at the insertion of the rhomboids into the scapula, at the insertion of the deltoid into the humerus, at the insertion of the sacrospinalis, and along the crest of the ilium and at the insertion of the *gluteus medius*. The most effective form of treatment by physical means is as follows:

1. Obtain relaxation of the associated muscle spasm by heat—radiant heat, infra-red irradiation or short-wave therapy.

2. Search for these myalgic spots and disperse them by injecting ten to twenty mils of "Novocain" into the area. As soon as anæsthesia is present follow this operation by deep heavy massage.

3. Give appropriate exercises, to put the muscles through as full a range of movement as possible. Injections may have to be repeated, as when the main spot is removed, other smaller spots in the vicinity may be found. These are dealt with in the same way.

Neuralgia and Neuritis.

The principal factor to be watched in treating by physical therapy neuralgia and neuritis, including brachial neuritis and sciatica, is to use very low intensities for short intervals in the early stages. Anything beyond the gentlest warmth will aggravate the pain. The explanation of this is probably that any increase in tension in a nerve already inflamed and enclosed in a tough sheath increases pain.

Bell's Palsy.

In Bell's palsy short-wave therapy, should be applied with one small electrode at the site of the exit of the nerve onto the cheek. Infra-red irradiation with the localizer gives equally good results. This should be combined with massage to help keep tone in the muscles, and exercises as soon as recovery commences. Splinting of the mouth is necessary.

Low Back Pain.

In a talk such as this time will permit of only a brief enumeration of the less often looked for causes of low back pain. The vast majority of cases are postural in origin; after examining the back, always look at the feet.

1. Flat foot with eversion. Eversion of the feet tends to rotate the femur outward, with resultant displacement of the greater trochanter backward. To compensate for this, there is a greater degree of lordosis with increase of muscle strain and backache.

2. Osteoarthritis of the feet or knees. Faulty posture with back strain again occurs in this condition. The patient is trying to get a comfortable position for the foot or knee and throws added strain on the muscles and ligaments of the spine.

3. Shortened hamstrings. This condition is one of the most frequent causes of backache among soldiers in hospital. It is stated that the centre of gravity of the body is altered by the weight of the pack and equipment. The explanation of why the condition appears to be more prevalent in military than in civil life may be that the civilian says he has a "touch of rheumatism" and proceeds to swallow various advertised remedies and help swell the millions spent per year in Australia on patent medicines. This condition is frequently post-operative, when a patient has spent some time propped up in bed with a pillow under the knees.

4. Shop assistant's back. People who stand for long periods tend to assume a stance with one knee hyper-extended and the other knee flexed. The result is a tilting of the pelvis, which tends to a permanent scoliosis with resultant pain.

5. Motor-car driver's back. Backache occurs in motor-car drivers if the seat is not adjusted to give support under the thighs. This condition is becoming increasingly frequent among the female drivers of army vehicles, ambulances *et cetera*. The condition is relieved by raising the front of the car seat so that support is given to the whole length of the thighs.

6. Prostatitis and vesiculitis. There is now a greater realization that a low-grade infection of the prostate, even without urinary symptoms, can cause backache. Drainage is increased by heating with the diathermy electrode, and if this is followed by prostatic massage, the infection is cleared and backache is relieved.

7. Slipping of the sacro-iliac joints. Place the patient on the side with the affected joint uppermost; flex his upper leg with the toe behind the knee of the other leg. Place the palm of the hand on the joint and the other on the front of the shoulder. By pushing with the hands in opposite directions the body is rotated strongly.

Arthritis.

The subject of arthritis is too vast for me to give anything but the briefest outline of the different forms of physical treatment employed. First of all, any of the following conditions that may be present should receive attention: sepsis, obesity, menopause, constipation, achlorhydria *et cetera*. Physical treatment consists of a combination of five basic principles—heat, the wax bath, exercises, counter irritation and occupational therapy.

1. For local heating of joints in general, use short-wave therapy for deep-seated joints covered by much muscle, such as the hip, spine, shoulder *et cetera*. For more superficial joints, such as the foot, knee, elbow *et cetera*, a more intense erythema can be obtained by infra-red irradiation.

2. A useful means of treating the hands in rheumatoid arthritis is by means of the paraffin wax bath. Wax with a melting point of 120° F. should be used. The hands are dipped into the wax repeatedly, and after each immersion the wax is allowed to harden till a thick glove covers the hands. This glove is left on for about fifteen minutes.

3. Counter-irritation of joints is best obtained by either hystamine or "Mercolyl" iontophoresis. A useful method of applying histamine is to use a 2% histamine ointment, which is rubbed well into the area and then covered with moist gauze under the tinfoil plate attached to the positive plate of a galvanic machine. The negative plate can be placed on any convenient part of the body. A current of two or three milliamperes for ten minutes will produce an intense hyperæmia with weal formation.

4. Exercises. Appropriate exercises for each joint are then given to increase the range of movement.

5. Occupational therapy. A great deal can be done for these sufferers by occupational therapy. They receive a mental stimulus as well as exercising the appropriate joints. The use of foot and hand looms for weaving, basket and leather work, pottery, carpentry *et cetera* are extremely beneficial. These patients grow tired of purposeless exercises, but will exercise joints if they can be producing something at the same time.

Gonorrhœal Arthritis.

Artificial fever is an absolute specific for gonorrhœal arthritis. It fulfils all the requirements of treating in one fell swoop the focus of infection and the joints involved. Artificial fever is also indicated in tabes, general paralysis of the insane, chorea, early disseminated sclerosis and certain forms of infective arthritis. Fever up to 105° F. is induced and maintained in sessions of four to eight hours.

HIRST'S HÆMAGGLUTINATION PHENOMENON EXHIBITED BY RICKETTSIA ORIENTALIS (SYN. TSUTSUGAMUSHI).

By J. L. O'CONNOR,

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SINCE Hirst (1942) first described the phenomenon of agglutination of fowl's red cells by influenza virus suspensions and demonstrated its value as a means of estimating the amount of virus present in a suspension, many workers have reported that other viruses have possessed the same property of agglutinating fowl's red cells. Clark and Nagler in 1943 published the results of investigations into the susceptibility of various types of erythrocytes to agglutination by a number of different viruses. In connexion with the work to be described here, it is of particular interest to note that approximately only half of the samples of fowl's cells used by Clark and Nagler were agglutinable by vaccinia virus.

During the course of other investigations into the behaviour of *Rickettsia orientalis* it became important to have available a simple means of assessing potency of suspensions. The relation of this organism to the hæmagglutination phenomenon was therefore investigated.

It had been noticed at times, when membranes were being harvested, that the embryo's blood showed some slight tendency to clump on the membranes. It was thus anticipated that the reaction would be obtained *in vitro*.

Materials and Methods.

Source of Rickettsiæ.—Rickettsiæ were grown on the chorio-allantoic membrane of duck embryos, as first described by Gispen (1940). Lewthwaite, O'Connor and Williams had found that this species of Rickettsia failed to become adapted to the chorio-allantois of chick embryos. Fertile eggs from Indian Runner ducks were first incubated for fourteen days at 39° C. They were then inoculated with infective material (usually egg passage "Seerangayee" strain) after the chorio-allantoic membrane had been "dropped" by Burnet's method. Membranes were harvested after seven days' incubation at 35·5° C. They were then ground with sterile quartz sand and suspended in physiological saline solution to give a 10% suspension, which was then centrifuged in an angle centrifuge for five minutes to remove sand and debris. In two experiments described below the material was inoculated into the allantoic cavity and then incubated as usual.

Erythrocyte Suspensions.—Red cells were prepared in 2% suspensions in physiological saline solution containing calcium chloride (0·22%).

Specific Serum.—Serum from a patient convalescent from scrub typhus contracted in New Guinea, giving a positive agglutination response with Proteus OXK, was used. The serum was inactivated by heating in a water bath at 56° C. for thirty minutes. Only one specimen of serum was available; it was obtained through the kindness of Dr. E. H. Derrick, of Brisbane. The tests were set up according to the method described by Nagler (1942).

Range of Cells.

It was thought that duck's cells would be the most suitable for test. Unfortunately the only specimens of cells available were from Muscovy ducks. These are a different type from Indian Runners. Four specimens of cells were tested, but all gave negative results.

Fowl's cells were then tested. Samples were obtained from six fowls (all adult female White Leghorn birds). Four of these gave no agglutination. The Rickettsial suspension was obtained from ten membranes showing good growth that were pooled for the preparation of an experimental vaccine. The results are shown in Table I.

TABLE I.

Cells.	Initial Dilution of Suspension.							
	1/4	1/8	1/16	1/32	1/64	1/128	1/256	Control.
AX	++[1]	++	++	++	++	+[1]	±[1]	0
B.18	++	++	++	+	±	0[1]		0

[1] "++" = complete agglutination; "+" = partial agglutination; "±" = trace of agglutination; "0" = no agglutination.

Cells of fowls numbers 17, 19, 20 and 21 gave no agglutination at a dilution of one in four. Six samples of cells from day-old chicks all failed to give a positive result. One specimen of each of human, sheep and guinea-pig cells gave negative results. Pressure of other work has prevented a proper investigation of the range of susceptible cells.

Temperature and Time.

Reactions were tested at 37° C., at room temperature (20° C.) and in the refrigerator (6° C.). No distinguishable pattern was observable after two hours at 37° C. At room temperature and in the cold the reaction was complete at four hours and the pattern remained stable overnight.

Specific Serum Inhibition.

Rickettsia-serum mixtures were incubated at 37° C. for thirty minutes prior to the addition of red cells. A constant dilution of serum (1 in 4) was added to each dilution of rickettsial suspension. The results are shown in Table II.

Adsorption from Allantoic Fluid by Embryonic Duck's Erythrocytes.

Two experiments were performed to ascertain if adsorption from allantoic fluid onto embryonic erythrocytes and subsequently elution, as described by Francis and Salk (1942) for influenza virus, would occur.

Experiment 1.—Six eggs were inoculated in the allantoic cavity with egg-passage material, and after seven days' incubation they were opened through the air sac and the

TABLE II.

Cells.	Serum.	Dilution of Rickettsial Suspension. 1/2	1/4	1/8	1/16	1/32	1/64	1/128	1/256
AX	Convalescent	0[1]	0	0	0	0	0	0	0
	Normal	++[1]	++	++	++	++	+[1]	±[1]	0
	Nil	++	++	++	++	++	+	±	0
B.18	Convalescent	0	0	0	0	0	0	0	0
	Normal	++	++	++	+	0	0	0	0
	Nil	++	++	++	+	0	0	0	0

[1] "++" = complete agglutination; "+" = partial agglutination; "±" = trace of agglutination; "0" = no agglutination.

umbilical vessels were cut and allowed to bleed for two or three minutes into the allantoic fluid. The blood-stained fluid was collected and allowed to stand overnight in the refrigerator. On the following day the mixture was centrifuged and the deposit separated from the supernatant allantoic fluid. The deposit of red cells was washed twice with sterile saline solution and then resuspended in a volume of saline solution equal to the original volume of allantoic fluid. Tenfold serial dilutions in normal saline solution were then prepared of each of the supernatant allantoic fluid and the red-cell suspension. These were then injected intraperitoneally into mice in doses of 0·5 cubic centimetre. The results are shown in Table III. Calculated by the method of Reid and Muench, the "L.D.50" of the allantoic fluid supernatant was 1/150 and of the red cell suspension 1/4,800. In other words, 97% of the infective particles had been adsorbed onto the embryos' red cells.

TABLE III.

Dilution.	Supernatant Allantoic Fluid.	Red Cell Suspension.
10^{-2}	3/5[1]	5/5
10^{-3}	0/5	4/5
10^{-4}	0/5	2/5
10^{-5}	0/5	0/5

[1] The numerator indicates the number of deaths occurring in four weeks, and the denominator the total number of mice in the group.

Experiment 2.—At a later date the experiment was repeated. On this occasion no egg-adapted material was available for infecting the allantoic cavity, owing to difficulty in obtaining fertile duck eggs. Ascitic fluid from a guinea-pig dying of scrub typhus was used as inoculum. Allantoic fluid was harvested on the sixth day after the embryos were bled as described above. The mixture of blood and allantoic fluid was treated as described above for the previous experiment. The results are shown in Table IV. The "L.D.50" for the supernatant is 1/3 and for the red cell suspension 1/170 approximately, so that about 96·5% of the infective material from the fluid is contained in the red-cell suspension.

TABLE IV.

Dilution.	Supernatant Allantoic Fluid.	Red Cell Suspension.
None	5/5	5/5
10^{-1}	0/5	5/5
10^{-2}	0/5	2/5
10^{-3}	0/5	2/5
10^{-4}	0/5	0/5

Comment.—Centrifuging was carried out in an angle centrifuge at 2,000 revolutions per minute for five minutes only. Our constant experience has been that this does not deposit any detectable number of Rickettsiæ. Elution of Rickettsiæ from the red cells was attempted on both occasions by treatment in the water bath for two and a half hours at 37° C. In neither case was any evidence of elution obtained.

Summary.

1. *Rickettsia orientalis* grown in the duck egg has been shown to give Hirst's hæmagglutination phenomenon.

2. Serum from a patient recovered from scrub typhus contains specific inhibiting antibodies.

3. Embryonic duck's erythrocytes possess the power of adsorbing most of the infective agent from allantoic fluid.

4. The property of agglutination with *Rickettsia orientalis* was found in only two samples of fowl's cells out of a total of eight tested. None of six samples from day-old chicks gave agglutination.

5. One sample of each of human, sheep's and guinea-pig's cells lacked the property of agglutination with *Rickettsia orientalis.*

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A SYNDROME IN VIVAX MALARIA PROBABLY DUE TO SPONTANEOUS SUBCAPSULAR HÆMORRHAGE OF THE SPLEEN.

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THE syndrome to be described in this report, although occurring infrequently, is worthy of notice, since it may mimic serious acute thoracic and acute abdominal conditions, and unnecessary surgical measures may be undertaken for a condition which resolves satisfactorily under conservative treatment. The available standard textbooks on tropical diseases do not mention the syndrome, although traumatic and spontaneous rupture of the spleen in malaria have long been recognized.

Many service personnel, both during the war and for some years afterwards, will suffer recurring attacks of vivax malaria, unless a new drug is found which will cure the disease—a prospect which seems unlikely in the near future. Consequently this syndrome will be widely seen and may cause considerable worry to any army or civilian practitioner who should encounter a case, if its nature is unrecognized.

THE SYNDROME.

The syndrome is characterized by the dramatically sudden onset of severe pain in the left upper quadrant of the abdomen, made worse by breathing, and often referred to the tip of the left shoulder. Tenderness is present in the left hypochondrium. There may be rigidity of the upper part of the abdomen, and the pain, which is constant, lasts for a few days or even many weeks. This occurrence is usually closely related to an overt attack of vivax malaria, coming on soon after the onset of fever, but in some cases, days or even many weeks later. It is entirely unrelated to trauma. More than one attack may occur, and complications may develop at the base of the left lung.

Incidence.

Table I sets out the ten cases of the syndrome observed in this hospital from April to September, 1944. In the six months, 5,822 patients suffering from proved vivax malaria were admitted to hospital and treated. None of these men were reexposed to malaria infection during that time. In the same period, 216 patients with unconfirmed vivax malaria were treated. The total is thus 6,038—an incidence of one in about 600. Only twelve cases of falciparum malaria were diagnosed in these six months.

TABLE I.

Case Number.	Relation of First Hæmorrhage to Malaria.	Number of Previous Attacks of Malaria.	Period Out of New Guinea.
I	Fourth day of therapy (QAP)[1]	Nil.	2 months.
II	Ninth day of "Atebrin" maintenance course (following QAP)	Nil.	2 months.
III	Second day of therapy (QAP)	Nil.	2 months.
IV	First day of therapy (QAP)	5	3 months.
V	Third day of "Atebrin" maintenance course (following QAP)	1	3 months.
VI	Second day of illness (before therapy started)	1	2 months.
VII	Third day of illness (before therapy started)	Nil.	4 months.
VIII	Second day of illness (before therapy started)	Nil.	2 months.
IX	Ninth day of therapy (QAP)	1	6 months.
X	Twelve weeks after an attack of malaria treated with QAP	3	6 months.

[1] QAP = Quinine (three days), "Atebrin" (five days), "Plasmoquine" (three days), in sequence.

Onset.

In all cases the onset was marked by the sudden occurrence of pain. Six patients were lying in bed; one was awakened by pain, two were sitting down, and one was stricken in the act of swallowing beer. There was no evidence of injury in any case, either at the time or in the immediate past.

The onset occurred in three cases, before treatment started: in four cases, during treatment; in two cases, early in the six weeks' maintenance course of "Atebrin" therapy; and in one case, three months after an attack of malaria.

The onset occurred at this hospital in six cases—at a casualty clearing station in two cases, and in two cases at the patients' units. Those who became ill elsewhere than here were admitted to hospital within a day or two.

Five had a history of previous malaria; five had none.

Table II gives the essential clinical features.

Symptoms.

The pain was always severe, and in some cases, very severe. The appearance of those who suffered from the more severe degrees of pain superficially resembled that of patients in a state of surgical shock. The demeanour was anxious and alert, the skin was pale and the pulse was rapid. The blood pressure of one patient who was examined a few minutes after the onset was 120 millimetres of mercury (systolic) and 80 millimetres (diastolic).

It can be seen from Table II that all complained of pain in the left upper quadrant of the abdomen. In four cases the pain was also severe in the epigastrium, and in six cases pain was referred to the left shoulder, to the tip of the acromial process. It was made worse by deep breathing in all cases except one (Case IV); this patient had no shoulder pain, and suffered the mildest attack, which was of the shortest duration. All patients stated that the pain was constant, except for its accentuation on deep breathing, and that it gradually decreased in intensity, until pain was complained of only on deep breathing or coughing. The shoulder pain generally abated with the abdominal pain, although in a few cases the abdominal pain lasted longer.

Signs.

Only two signs were recognized in the condition: first, tenderness in the upper part of the abdomen, always in the left hypochondrium, combined sometimes with tenderness in the epigastrium; and secondly, a varying degree of muscular guarding, which, shortly after the onset, appeared to be more "voluntary" than the reflex rigidity discovered in the presence of a perforated viscus. An enlarged spleen was palpated in six cases, but tenderness and rigidity may prevent accurate palpation in the early stages.

Fever, apart from that of still active malaria, formed no part of the syndrome, nor was the pulse rate raised except in the first hour or two of very severe pain.

Laboratory Data.

No detailed hæmatological studies were undertaken. The blood of the patient in Case VI, two days before his third attack (Table II), had a hæmoglobin value of 13·3 grammes *per centum*; his red cells numbered 4,140,000 per cubic millimetre. Two days after the onset of pain the hæmoglobin value was 14·3 grammes *per centum*, and three days after this the hæmoglobin value was 13·8 grammes *per centum* and the red cells numbered 4,770,000 per cubic millimetre. The hæmoglobin value in Case VII was 15·9 grammes *per centum* one hour after the onset. Twenty-four hours later it was the same.

The few white blood cell counts gave essentially normal results—both total cell counts and differential counts (Table II).

X-Ray Appearances.

An X-ray examination of the lungs was made in eight cases. In the other two cases there was no clinical suggestion of any pulmonary disease. In three of the eight, congestive changes were seen, and in one of these a small collection of fluid was apparent.

In Case II, three days after the onset, a small, well-defined area of congestion was found in the middle zone of the lower lobe of the left lung. Seventeen days later these radiological signs had almost completely resolved. In this case some clinical signs of early consolidation developed on the second day at the base of the left lung.

In Case III the clinical signs were similar on the second day after the onset. In addition, a friction rub was heard at the base of the left lung posteriorly. An X-ray photograph on the third day revealed congestion at the base of the left lung, with a small amount of fluid; the left side of the diaphragm was slightly raised. On the twenty-third day the lung fields were radiologically clear except for some adhesions at the left costophrenic angle.

The changes were similar, but more apparent in Case VI. Five days after onset there were radiological signs of congestion at the base of the left lung, and seventeen days later the left side of the diaphragm was seen to be raised. On the forty-third day after the initial onset a second attack occurred. On this day a friction rub was heard at the base of the left lung posteriorly, and radiological signs of congestion reappeared. Five days later a third sudden attack of pain in the left side of the chest occurred and was followed in four days by the onset of pneumonia at the base of the left lung; extensive consolidation was apparent, clinically and radiologically. This patient was evacuated to a base hospital fifteen weeks after the onset. Six months from the original attack, radiological signs of an unresolved parenchymal lesion were still present at the base of the left lung.

The area of congestion in all three cases was not at the extreme base, but appeared in the X-ray photographs just to the left of, and continuous with, the shadow of the upper left ventricular region. The congestion observed in the X-ray films cannot be given any certain pathological interpretation. Because of the severe pain and consequent splinting of the diaphragm, it seems likely that some collapse of the lung may occur. Alternatively, the clinical and radiological signs could be interpreted as hypostatic congestion from the same cause.

TABLE II.

Case Number.	Age. (Years.)	Symptoms.	Site of Pain.	Duration of Pain.	Number of Hæmorrhages.	Signs.	Initial X-Ray Examination of Chest.	Initial Number of Leucocytes per Cubic Millimetre.
I	39	Severe abdominal pain, worse on breathing.	Left hypochondrium and tip of left shoulder.	7 days.	1	Spleen enlarged two fingers' breadth below costal margin, very tender, some rigidity.	Not made.	Not estimated.
II	29	Very severe abdominal pain, worse on breathing.	Epigastrium, left hypochondrium and tip of left shoulder.	11 days.	1	Rigid and very tender left upper quadrant of abdomen. Signs at base of left lung.	Congestion, base of left lung (2nd day).	10,200 (Neutrophile cells 40%) 4th day.
III	24	Very severe abdominal pain, worse on breathing.	Epigastrium, left hypochondrium, tip of left shoulder.	4 weeks.	1	Spleen enlarged one finger's breath below costal margin, and tender before onset. After onset, rigid and tender left hypochondrium. Pale and "shocked". Friction rub, base of left lung on third day.	No abnormality detected (5th day). Fluid and congestion of base of left lung (11th day).	10,200 (Neutrophile cells 68%) 3rd day.[1]
IV	25	Severe abdominal pain.	Left hypochondrium.	2 days.	1	Spleen enlarged two fingers' breadth below costal margin and very tender. Some rigidity of left hypochondrium.	No abnormality detected (6th day).	7,600 (Neutrophile cells 57%) 4th day.
V	34	Severe abdominal and shoulder pain, worse on breathing.	Left hypochondrium and left infraclavicular fossa.	14 days.	1	Tender left hypochondrium.	No abnormality detected (8th day).	11,000 (Neutrophile cells 46%) 6th day.
VI	26	Very severe abdomen, back and shoulder pain, worse on breathing.	Epigastrium, left hypochondrium, left costo - vertebral angle and tip of left shoulder.	14 weeks. (Evacuated to base.)	3	Spleen enlarged one finger's breadth below costal margin before onset. Tender left hypochondrium. Signs at base of left lung on third day. Later pneumonia, base of left lung.	Congestion, base of left lung (6th day).	8,600 (Neutrophile cells 63%) 5th day.
VII	40	Severe abdominal pain, worse on breathing.	Left hypochondrium.	3 weeks.	1	Tender left hypochondrium.	Not made.	7,600 1st day.
VIII	23	Severe abdominal pain, worse on breathing.	Left hypochondrium, tip left shoulder.	16 days.	2	Tender left hypochondrium.	No abnormality detected (3rd day).	3,400 (Neutrophile cells 24%) 2nd day.
IX	36	Severe abdominal pain, worse on breathing.	Left hypochondrium, tip left shoulder.	12 days.	1	Spleen enlarged two fingers' breadth below costal margin and very tender.	Not made.	Not estimated.
X	21	Severe abdominal pain, worse on breathing.	Epigastrium and left hypochondrium.	4 weeks.	1	Spleen enlarged one finger's breadth below costal margin and tender; rigid in left hypochondrium.	No abnormality detected (15th day).	10,300 (Neutrophile cells 72%) 11th day.

[1] Furuncle of the chest, with axillary adenitis.

Complications.

The only complication observed was congestion at the base of the left lung, which proceeded in one case to pneumonic consolidation (Case VI). This patient, four days after his third attack, had fever, rapid respiration and a rise in pulse rate. The white blood cells numbered 18,200 per cubic millimetre, and the diifferential count gave the following results: neutrophile cells, 78·5%; lymphocytes, 17·5%; monocytes, 4%. Culture from sputum yielded a moderate growth of pneumococcal colonies (not typed), with a few hæmolytic streptococci. The response to sulphamerazine was satisfactory, and the patient was afebrile three days after its exhibition.

Spontaneous rupture was not observed.

Diagnosis.

In a patient suffering from an attack of vivax malaria, or recently recovered therefrom, the sudden onset of severe epigastric and left-sided upper abdominal pain, referred to the left shoulder and made worse by deep breathing, in the absence of trauma, is most likely to be an example of this syndrome.

Emphasis in the diagnosis is placed on the abruptness of onset and on the severity of the pain. Tenderness and some voluntary rigidity are present in the left upper quadrant of the abdomen. The finding of an enlarged and tender spleen is of considerable help in the diagnosis, but it is probably unwise to search too diligently for a spleen suspected to be the seat of hæmorrhage.

There is no rise in temperature, unless overt malaria coexists, and only a slight rise in pulse and respiratory rates. The total and differential leucocyte counts are essentially normal.

Differential Diagnosis.

From the description given, it is apparent that a number of acute upper abdominal and acute left thoracic conditions may be simulated.

Spontaneous Rupture of the Spleen.—The spleen may rupture, flooding the abdomen, or delayed rupture may occur, such as is known to the French as "*hémorragie en deux temps*". It may be as difficult as it is important to decide whether intraperitoneal hæmorrhage has occurred, since the symptoms and signs in either case may be similar if a subcapsular hæmorrhage is large or an intraperitoneal hæmorrhage is small. Loss of blood, which is limited in a capsular hæmorrhage, is usually severe in rupture, and considerable shock, persisting pallor, a rise in the pulse rate, falling blood pressure and a progressive fall in hæmoglobin readings, with signs of hæmodilution, indicate hæmorrhage into the abdominal cavity. Hæmoglobin estimations and hæmatocrit readings soon after the onset and repeated every day or more frequently should therefore be made if there is any doubt.

Perforated Peptic Ulcer.—The pain in some cases of perforated ulcer may be in the epigastrium and left hypochondrium and referred to the left shoulder. It usually spreads speedily over the whole abdomen. Vomiting is common. The resulting rigidity is more constantly found and board-like in character, and the tenderness is more diffuse, than in the syndrome under discussion. Liver dulness may be absent.

Spontaneous Pneumothorax (Left).—The onset of the syndrome under discussion may mimic this spontaneous left-sided pneumothorax, but careful examination of the lungs, the position of the trachea and the apex beat should

exclude the condition. Cyanosis and a raised respiratory rate are usual.

Left Basal Pneumonia.—The onset in lobar pneumonia may be sudden, but it is seldom quite so dramatic as in the syndrome under discussion. However, this abrupt initial period of pain is sometimes observed. At this stage headache is present and often a rigor occurs, and the temperature and pulse and respiratory rates rise. These symptoms are associated with a malarial paroxysm; parasites, however, are usually found in the blood. Cough with sputum and a neutrophile leucocytosis would be a strong indication of pneumonia. The early onset of left basal pulmonary signs, which follow in some cases of the syndrome, may easily obscure the true diagnosis. However, rusty sputum, rising fever and increasing pulse and respiratory rates with a neutrophile leucocytosis should make the diagnosis clear.

Diaphragmatic Pleurisy and Epidemic Pleurodynia.—The main points in pleurisy which differentiate it from the syndrome are fever, absence of tenderness (or only moderate tenderness in the abdomen, even if some reflex rigidity is present), and the common radiological finding of a pulmonary tuberculous focus. Moreover, the pain in pleurisy has not usually the sudden onset or the constant quality of the syndrome. Recently, epidemic pleurodynia, with increased pain on breathing, has in this area given rise to considerable difficulties in diagnosis when the pain is on the left side, although right-sided pain is more common. However, the pain is not so severe or so sudden at onset, nor has it such a constant quality. Malaise, headache and fever accompany the onset. A friction rub develops in some cases and adds to diagnostic difficulties. Only one patient with epidemic pleurodynia, of 35 patients examined, complained of shoulder pain. Leucocytosis may or may not be present. This epidemic did not start until two months after the last case of the syndrome in this series.

Miscellaneous.—Acute pancreatitis, coronary thrombosis and ectopic pregnancy may need to be considered.

Management.

Once the syndrome is diagnosed, morphine is indicated for the pain. A leucocyte count should be made, and the chest radiologically examined if pulmonary signs develop. As has been indicated previously, a hæmoglobin estimation, erythrocyte count and hæmatocrit reading should be made, and repeated, if there is any doubt about the occurrence of hæmorrhage into the peritoneal cavity.

In all the cases reported here breathing exercises were given, a start being made a few days from the onset. The wisdom of this measure is not certain. One wishes to prevent collapse of the base of the left lung, but full ventilation may possibly increase the extent of a subcapsular hæmorrhage, assumed here to be the cause of the syndrome, or may conceivably make delayed rupture more likely.

If delayed rupture does not occur, and if there is no infection in the collapsed lung, the prognosis for full recovery is excellent. It seems reasonable to give the patient a considerable period of convalescence, depending on the degree of severity and the time taken for recovery from the pain. Too early return to the unit, except when the patient's occupation is sedentary, may lead to rupture of the spleen from even minor trauma.

Comment.

The cause of this syndrome is not yet known with certainty. Operative interference would in all probability reveal the gross pathological condition, but no patient in this series has been subjected to operation. It would be of considerable interest to know the findings at operation on any patients who have presented this syndrome and been submitted to abdominal section.

It is likely that only three conditions could cause this syndrome: first, a small and limited spontaneous rupture of the spleen; secondly, a splenic infarct; and thirdly, a spontaneous subcapsular hæmorrhage of the spleen.

1. The syndrome is similar to that of spontaneous rupture of the spleen. However, in this condition commonly abdominal pain is by no means prominent. Moreover, the pain often lacks the initial sudden onset, which was noted in all these cases. It is probable that minor degrees of rupture may occur which will resolve without surgical treatment, although in cases in which conservative treatment is given the question of whether rupture has in fact occurred must remain speculative. But it seems unlikely that ten consecutive cases of rupture of the spleen should occur, and that all the patients should recover without operative treatment. The blood findings already mentioned in Case VI, which were the most severe, did not suggest that a hæmorrhage of any size had occurred. This was true also of Case VII.

2. It is more difficult to refute the occurrence of a splenic infarct as the cause of the condition. Infarcts do occur in malaria and in many other diseases; but it is common knowledge that autopsy findings show that these frequently occur with no sign or symptom to indicate their presence *ante mortem*. Possibly the ten cases in this series represent the only infarcts which occurred in the 6,038 cases of vivax malaria, and which gave rise to signs and symptoms. However, the shoulder pain (of common occurrence) and the train of symptoms which arose in the pleural sac and base of the left lung seem unlikely to follow an infarct of the spleen, and have not in fact been recorded.

3. Subcapsular hæmorrhage of the spleen appears to be the most likely explanation for the syndrome. The signs and symptoms are those which one would expect to follow such a vascular accident. The sudden stretching of the capsule as the result of hæmorrhage would give rise to abdominal pain and tenderness, while irritation of the diaphragm would, through afferents in the phrenic nerve, cause shoulder-tip pain. Moreover, as the result of continued pain and irritation and consequently the restriction of normal diaphragmatic movement, it is not difficult to understand the changes that occur in the left lung. At autopsies in Greece performed on subjects of chronic malaria, N. Hamilton Fairley has observed large calcified plaques partly replacing the capsule of the spleen (personal communication). He has suggested that these represent the end-result of a subcapsular hæmorrhage.

One patient (Case X) was examined five months after the onset. He had been taking "Atebrin" (0·7 gramme per week) since the attack. He complained of some aching in the left hypochondrium after severe exercise. The spleen was still enlarged one finger's breadth below the left costal margin and was tender. X-ray examination of the spleen, however, failed to reveal any evidence of calcification. If it is practicable, some of these patients will be radiologically examined at intervals.

The mechanism can only be guessed at. Possibly in the splenic arteries of these subjects, degenerative changes occur and predispose the subject to such a vascular accident. Susman states that in 50% of spleens, by the age of thirty-six years degenerative changes are present in the vessels. It is certain, however, that in some way the intense activity of the spleen in malarial infections must play a part in the causation, whether from congestion, stasis, phagocytosis, fibrosis, damage to endothelium from liberated toxins, or a combination of some or all of these factors.

It is probable that the initial hæmorrhage is in the substance of the spleen, and that the blood finds its way outwards under the capsule to produce the severe pain complained of and the tenderness found on examination. When a large hæmorrhage occurs, the capsule is involved over a greater area, more afferents are stimulated, and the pain is greater and lasts longer. In this case the chance of delayed rupture must be greater than when the hæmorrhage is small.

It is not suggested that this syndrome is peculiar to vivax malaria, but merely that the patients observed were known to have had a vivax infection. It may well be that it occurs with falciparum malaria, and, in retrospect, I can remember in New Guinea a few cases in which an overt falciparum infection was present.

In conclusion, it is suggested that the available evidence points to a subcapsular hæmorrhage of the spleen as the most likely cause of the condition. Whatever the mechanism may be, there is no doubt that the syndrome can be readily recognized, that conservative treatment can be instituted, and that a favourable prognosis can be given.

SUMMARY.

1. Ten cases of an acute abdominal syndrome in vivax malaria are described.
2. The syndrome is characterized by sudden severe pain in the left upper quarter of the abdomen, usually referred to the tip of the left shoulder; it is accentuated by deep breathing, and lasts for periods varying from a few days to many weeks. Tenderness is present in the left hypochondrium, but no true rigidity.
3. The onset of the syndrome was in no case related to trauma. It is usually closely related to an overt attack of malaria, but may occur three months later. In two cases more than one attack occurred.
4. The incidence was about one in each 600 treated attacks of vivax malaria.
5. Changes were found at the base of the left lung in three cases; friction rub, a small pleural effusion, signs of congestion with radiological shadows, and complicating pneumococcal pneumonia were all observed.
6. All patients recovered with conservative treatment.
7. Spontaneous subcapsular hæmorrhage of the spleen is thought to be the most likely cause of the syndrome.

ACKNOWLEDGEMENT.

I wish to thank the Director-General of Medical Services for permission to publish this paper.

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HÆMOPERICARDIUM AS AN AUTOPSY FINDING.

By C. DUNCAN,
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HÆMOPERICARDIUM—the presence of blood or blood-stained fluid in the pericardial cavity—is a subject which is almost neglected in most text-books on pathology and medicine. Some books dismiss the problem in a few lines; in others, the name does not even appear in the index. During the last nine years this condition has often been observed at autopsy examinations—so often that a brief summary of the frequency and causes was considered worthy of record.

Observed Frequency.

Since 1936 I have carried out 1,108 autopsy examinations in southern Tasmania. Of these, 413 were requested by public hospitals or private practitioners, and 695 were performed at the direction of a coroner. The latter group includes deaths from unnatural causes (340 cases), and also deaths later found to be from natural causes but for which no medical practitioner was able to give a satisfactory certificate. In these 1,108 consecutive autopsies, hæmopericardium was observed on 36 occasions—3·25% (p.e.P. ± 0·35%)—so it cannot be regarded as a rare condition. This series includes one case in which pericarditis was also present.

Pathological Appearance.

The effusions could be divided into two types, as follows.

1. Simple blood clot. This was almost invariably produced by some mechanical process—leaking aneurysm, cardiac infarction or trauma. In this type the clot could normally be seen before the parietal section of the pericardium was opened. (A unique example of this type was seen in Melbourne some years ago. A radium needle had been inserted for breast carcinoma. The needle perforated the parietal section of the pericardium and scratched an elongated track through the visceral part of the pericardium and myocardium.)
2. A blood-stained effusion without clotting. This type was seen in effusions due to tuberculosis, neoplasms, blood disease and septicæmia.

Analysis of Causative Factors with Typical Cases.

Dissecting Aneurysm of the Aorta.

The nine cases of dissecting aneurysm of the aorta all occurred among elderly people, with generalized arteriosclerosis. In one case syphilis was also indicated.

Autopsy C175.—L.M., a male subject, aged sixty-five years, called out, vomited, and died one hour later. At autopsy, generalized arteriosclerosis was found. The pericardial sac was filled with several ounces of firm, dark blood clot, which had arisen from a dissecting aneurysm of the aorta situated three centimetres above the aortic valves. The aorta was grossly atheromatous, and the heart was moderately enlarged.

Cardiac Infarction.

The seven cases of cardiac infarction all occurred among people aged over fifty years. In all, occlusion of one or more main branches of the coronary arteries was present, with either partial or complete rupture of an infarcted area of heart muscle. The lower half of the left ventricle was the commonest site of rupture found. Syphilis was not found in any case.

Autopsy C242.—A.K., a male subject, aged seventy-three years, had complained of "indigestion" for several days. He was found dead in bed. At autopsy, generalized arteriosclerosis was present. Several ounces of firm blood clot surrounded the heart. In the right and left coronary arteries recent thrombosis was evident, and there was an area of cardiac infarction with partial rupture of the lower part of the left ventricle.

Mechanical Trauma.

In 12 of the 15 cases of mechanical trauma there was a crush injury of the chest.

Autopsy C190.—L.E.R., a male subject, aged twenty-six years, was thrown from a side-car following a collision, and was dead when picked up. At autopsy, bruises and abrasions to the head, arms, legs and chest, particularly over the centre of the sternum, were present. The left lung was perforated, and hæmothorax was observed. The liver and spleen were ruptured. The left radius and ulna were fractured, the left eighth and ninth ribs were fractured laterally, and there was a fracture through the centre of the sternum. The apex of the right ventricle was completely ruptured, extensive hæmopericardium having resulted. The parietal section of the pericardium was intact.

Autopsy C33.—W.J.W., a male subject, aged twenty years, was crushed by the wheel of a baker's cart. At autopsy were found bruising of the left knee, considerable surgical emphysema of the upper part of the chest, a fracture of the sternum in the central region, and dislocations of the fifth to ninth ribs on the right side. Bilateral pulmonary tuberculosis was also present. The pericardial sac was opened; the right and left ventricles were extensively torn, but little bleeding had occurred into the pericardial sac, owing to the rapidity of death.

Three cases were due to gunshot wounds of the chest, the pericardium only being perforated in one instance.

Autopsy C185.—M.H., a male subject, aged forty-five years, was found dead in his house. At autopsy a small-calibre bullet wound was present over the right eyebrow; the base of the skull was fractured in the anterior cranial fossa and the brain was bruised but not penetrated. A shot-gun wound was present in the left side of the chest just above the nipple, and the fourth and fifth left ribs were fractured. The left lung was perforated. Examination of the heart disclosed wide tearing of the left ventricle and a small amount of blood clot in the remains of the pericardial cavity.

This man appeared to have two mortal wounds, and murder was at first suspected. Autopsy findings and other evidence proved it to be a case of suicide.

Autopsy C610.—N.R.L., a male subject, aged sixteen years, was accidentally shot by a companion. He died about one hour later. At autopsy a bullet wound was found in the

left side of the chest. The 0·22 calibre bullet had passed between the second and third left ribs, perforated the margin of the left lung and the outer part of the parietal section of the pericardium, and buried itself in the spine. The heart was not injured; the pericardial sac contained a small amount of blood clot.

Secondary Carcinoma.

In the two cases of secondary carcinoma, metastatic invasion of the pericardium was present.

Autopsy H264.—W.H., a male subject, aged fifty-six years, was found at autopsy to have a carcinoma of the right lung arising from the right main bronchus; metastatic spread had occurred to the heart only. The visceral part of the pericardium was studded with miliary carcinoma. The pericardial sac was distended with about twenty ounces of blood-stained fluid.

Autopsy H402.—E.B.W., a male subject, aged thirty-four years, was found at autopsy to have a bronchiogenic carcinoma of the left lung with invasion of the pleural surface. Several pints of blood-stained fluid were present in the left pleural cavity. Early pneumonia was observed in the right lung, and metastases were found in the right lung and hilar lymph glands. The pericardial sac contained about fifteen ounces of deeply blood-stained fluid with some clot. The heart was slightly enlarged and the right ventricle was dilated. The visceral portion of the pericardium was dark red in colour and contained many raised, red, "vegetation-like" masses, as well as white nodules.

Tuberculous Pericarditis.

There was one case of tuberculous pericarditis.

Autopsy H125.—F.M., a male subject, aged fourteen years, was found at autopsy to have miliary tubercles in the lungs and spleen, tuberculous ulcers in the small intestine, and tuberculous peritonitis. The pericardial membranes almost filled the anterior aspect of the chest cavity. The parietal section of the pericardium was six to eight millimetres thick, not caseous, and adherent in places to the visceral part of the pericardium. About ten ounces of blood-stained fluid were found in the pericardial cavity. The heart was not enlarged. (See Figure I).

FIGURE I.
Tuberculous pericarditis. My = myocardium; V.P. = visceral pericardium; P.P. = parietal pericardium. (Five-sixths original size.)

Purpura.

There was one case of purpura.

Autopsy H316.—D.K. was a male subject, aged five years. At autopsy, bruises were found all over the body. The intestines were filled with blood, and hæmothorax was present. The pericardial cavity contained about five ounces of blood-stained fluid. The heart appeared normal.

Gas-Gangrene Septicæmia.

There was one case of gas-gangrene septicæmia.

Autopsy H124.—L.D., a male subject, aged forty years, had died of gas-gangrene septicæmia. At autopsy the heart was dilated, the heart muscle was soft and friable, and the endocardium was dark and evil-smelling. About four ounces of dark, blood-stained fluid were found in the pericardial cavity; *Bacillus welchii* was grown on culture from the fluid.

Summary.

In a series of 1,108 consecutive autopsy examinations hæmopericardium was observed on 36 occasions (3·25%). The factors found causing this condition are presented, with the findings in several autopsies.

Acknowledgement.

I wish to thank the Coroner, Colonel Clark, for permission to publish the findings in certain of the autopsies.

Reports of Cases.

A CASE OF SEVERE BURNS.

By H. B. HOLMES,
Surgeon Lieutenant, Royal Australian Naval Reserve.

THIS case is considered worth reporting, as it seemed remarkable that a man so extensively burned as this patient could recover. The area involved has been conservatively estimated at 70% of the body surface, but it may have been even more. The only parts not affected were the scalp, the feet, the upper three inches of both thighs, and one buttock.

Clinical Record.

R.S., aged twenty-five years, a stoker, was admitted to hospital at 9.15 a.m. approximately on January 5, after an accident in which a petrol engine that he was trying to start exploded. He was in great pain, but otherwise his general condition was good, probably because the accident had occurred only a few minutes previously, and his shock had not fully developed. He was immediately given morphine sulphate, 0·25 grain. Some enthusiast had already smeared him all over with grease. His temperature was 99·2° F., his pulse rate was 100 per minute, and his blood pressure was 150 millimetres of mercury (systolic) and 68 millimetres (diastolic). In view of this good condition it was decided to deal with his burns straight away.

The burns were mainly of the second degree, small areas of deeper burning being present on his hands and legs. The following areas were involved: the face, the neck, the shoulders, both arms and hands, the chest to the waist, the back to the waist, one buttock, and both legs from high on the thighs to the ankles.

He was lightly anæsthetized with "Pentothal", and the grease was removed by washing with soapy water, no brute force being used. In a few small areas ether had to be used, but it was used as little as possible. Such skin as was already loose was removed, but where the skin was not broken, the blisters were left undisturbed. The whole area was washed with saline solution, and then *tulle gras* dressings with a large combine dressing over the *tulle gras* were firmly bandaged in place. The whole procedure took about an hour. The intravenous administration of plasma was begun.

Prior to operation his hæmoglobin value, estimated by Sahli's method, was 92%. The administration of penicillin (15,000 units every three hours, with three grammes of sulphadiazine, followed by one gramme every four hours) was begun on his return from the operating theatre. Fluids were given freely by mouth, and all his urine was measured. In the next twenty-four hours he received four litres of serum, and his hæmoglobin value was 102%; it did not rise above 110% at any stage. His fluid intake by mouth was 152 ounces and his urinary output 58 ounces, the urine being normal. His loss of serum was most dramatically demonstrated when the serum soaked through the mattress and dripped onto the floor.

The intravenous administration of plasma was continued until his hæmoglobin value remained stationary at 90%;

this state was reached after four days, during which time he received a total of 12,000 mils of plasma. His dressings were left for nine days before they were changed, as his general condition, although as satisfactory as could be expected, did not permit of any unnecessary manœuvres. His temperature stayed at about 99·0° to 100° F., his pulse rate being 100 per minute, and his respirations numbering 20 to 22 per minute. He had "Carbogen" for five minutes at intervals of two hours. Slight green staining appeared on the dressings over his left arm and leg on the fourth day, and these areas were soaked in a 2% solution of acetic acid and the dressings were left. The administration of penicillin was continued, but that of sulphadiazine was stopped after a total of forty grammes had been given.

On January 15 (ten days after the accident) he was given morphine and his dressings were changed. Much of the area was healed and this was left exposed. His hands were healed except for some deep burns over the knuckles. His face, which had been treated with sterile "Vaseline" only, had healed except for the lips. His legs were infected with *Bacillus pyocyaneus* and had large sloughing areas. These sloughs were removed by gentle irrigation with saline solution, and then the parts were dressed with *tulle gras*, covered with large dressings soaked in acetic acid (2%).

After a visit by Colonel C. M. B. Littlejohn, the method of administration of penicillin was changed to that of a continuous intramuscular drip, 200,000 units being used in each 500 mils of saline solution in a "Soluvac" flask, and a fine hypodermic needle being employed as dripper. This proved much better while the man was ill; but once he recovered and tried to move about, he found that the needle in his muscles hindered his movements, and he asked for the three-hourly administration again. On the day following the changing of his dressings his general condition was much worse; his pulse rate was 140 per minute, his temperature was 102·6° F., and his respirations numbered 32 per minute. Examination of his chest revealed crepitations at the bases of both lungs. He was given three grammes of sulphadiazine, and then one gramme was given every four hours; an expectorant mixture was administered. This chest condition rapidly subsided.

On January 19 his dressings were changed; proflavine and glycerin (1/1,000) were used, and an immediate improvement in the appearance of his legs resulted. Culture of material from the areas yielded *Bacillus pyocyaneus, Bacillus proteus* (probably a contaminant), and a coliform bacillus. The hæmoglobin value was 75%, and the erythrocytes numbered 3,240,000 per cubic millimetre. He was given a transfusion of 1,100 mils of fresh blood by the intravenous drip method, and a blood count on the next day gave the following information: the hæmoglobin value was 90% and the erythrocytes numbered 4,400,000 per cubic millimetre. His legs were exposed to the sun every day for twenty to thirty minutes, and then redressed with proflavine and glycerin (1/1,000).

A slow but sure improvement occurred under this *régime*, and by February 6 no further dressings were needed on his legs, which had healed without grafting—a procedure which had seemed inevitable two weeks previously. He was allowed up under supervision by the physiotherapist and gradually regained his strength. His right hand, in which he had what was virtually a paralysis of the deep branch of his ulnar nerve, in that a palsy of the interossei and lumbrical muscles was present, took longest to recover full movement. He was sent south on convalescent leave on February 19.

Criticism.

I should like to make some self-criticisms. It would have been an improved treatment, in my opinion, if the patient had been given morphine intravenously for his original dressings, instead of being anæsthetized with "Pentothal", as this drug is detoxicated in the liver, and toxic liver damage is one of the late dangers of burns. An additional drug which could have been used to minimize the possibility of toxic damage to the suprarenals is cortical extract of some kind, but none was available in the area. In addition, the culture obtained from the infected areas is an extra indication, if one is needed, that penicillin inhibits the growth of Gram-positive organisms, but has no effect on the Gram-negative varieties.

Summary.

A case of burns is reported, in which full recovery occurred and in which the affected skin area covered 77% of the body surface (the figures mentioned by A. H. McIndoe in his articles in *The Lancet*, September 27, 1941) being used. Recovery took place in spite of some infection, which developed although prophylactic penicillin treatment was given. A logical treatment is described, with some criticisms.

Acknowledgements.

My thanks are due to the Director of Naval Medical Services for permission to publish this report, and to Colonel C. M. B. Littlejohn for his encouragement and advice.

A CASE OF FATAL STABBING BY A STINGRAY.

By R. J. WRIGHT-SMITH, M.D., F.R.A.C.P.,
Pathologist to the City Coroner, Melbourne; Pathologist, Royal Melbourne Hospital, Melbourne.

STINGRAYS, or stingarees, are fishes commonly found in Australian waters, and many examples of wounds caused by them have been recorded, some of them fatal. An excellent description of these fishes, their varieties, distribution and habits is given by Whitley (1940). He describes six varieties found in Victorian waters—namely, the smooth, sandy-backed, cross-backed, common, green-backed and eagle varieties. The black stingaree has, according to several fishermen, been caught in Port Phillip waters, but Whitley places this type in New South Wales waters only.

Whitley gives the following information:

> There are many species of stingrays or, as they are often called, stingarees. They are kite-shaped creatures and often have on the tail one or more serrated spines which can inflict a dangerous wound (*vide infra*). Stingrays, like their relatives, the skates, are excellent as food and can be recommended to invalids. Whereas skates lay eggs, the stingrays are viviparous. They live on the sandy bottoms of our beaches, harbours and continental shelf and even ascend rivers, feeding on any small fishes, worms, crabs and other creatures they may find there. The skin is not as rough as that of sharks and is not used for leather. The fins are used to make gelatine and the rest of the body makes good fertilizer. Natives of Pacific Islands use the spiny skin of some rays as a rasp (*Urogymnus*) or for drum membranes (*Himantura*), whilst the stings are used as needles or awls.
>
> The sting [Figure I] of the stingray forms a spur bound to the tail by strong ligaments and certain muscles which permit slight sideways movements, its length is variable. This armature is deciduous, it falls yearly, and another replaces it. If the shedding is irregular, two, three or even four spines may sometimes be found resting side by side. The spine is covered by a skinny sheath which is pushed back towards the base when the point penetrates the tissues of the wounded victim. The upper surface is convex, the lower surface

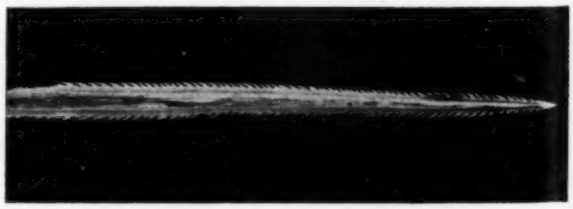

FIGURE I. The spine of a stingray.

> is scooped out into two lateral grooves situated on either side of a median ridge. These grooves shelter the venom glands. (According to some authors, the crack along the dorsal surface of the sting acts as a duct to convey the venom.) The edges of the spine are provided with serrations of which the barbs are directed towards the back; it also forms a spear resting on its lower surfaces in a gutter corresponding to the dorsal surface of the tail.

Muir Evans (1943) regards the serrated spine of the stingray as the first dorsal spine of a dorsal fin which has become obsolete. In other rays, two dorsal fins still remain, and like the spine, have been shifted onto the tail. He also regards the spine as an offensive or hunting weapon rather than as a weapon of defence. He describes in detail the venom apparatus. Cleland (1924, 1932) records several cases of wounds from stingray spines.

According to evidence given by an expert fisherman with long experience, stingrays captured in Port Phillip Bay have as many as four spines. A spine has been driven through the wood of a boat, and often the tail of the ray is broken and the spine projects from the end. The spines are not easy to see when the fish is swimming, as they lie flat on the tail. When the fish is excited, they are more or less erect. When attacked, the stingray shows great accuracy in stabbing while thrashing about, and it can protect the whole of its body from any direction. In the summer time, stingrays are common in Port Phillip waters and are especially seen in shallow water where they come for warmth. They lie in the sand, and if undisturbed, make no offensive action with the tail. Sometimes stingrays of large size are caught between sandbanks, and some stay there for a considerable time.

Clinical Record.

On January 9, 1945, Sergeant A.B., aged thirty-three years, a member of the Australian Military Forces, in company with other soldiers, proceeded to the Saint Kilda Baths for a swim. He entered the water at about 1.30 p.m. He was a powerful swimmer, and at about 2 p.m. was seen swimming strongly from the centre of the baths towards the landing steps. When almost to the steps, he disappeared below the surface and was seen to be waving his hands over his head. After a period of forty to sixty seconds he rose to the surface without properly breaking the water, and then sank again. His friends jumped into the water and assisted him up the steps. He was breathing in gulps, and it was noticed that he had a wound in his left breast, on the surface of which a drop of blood appeared. Artificial respiration was immediately performed. No bleeding from the wound occurred during this procedure. Life was pronounced extinct twenty minutes after removal of the patient from the water.

The water in the baths was dirty at the time, and nothing could be seen on the sea bed. The depth of the water was seven to eight feet at the site of the accident.

The palings of the fence enclosing the baths were broken in places, and it was possible for a fish to enter either through these gaps or beneath them when the sand was shifting. A careful examination of the sea bed, the steps and the piles revealed no weapon, spike or nail which could have caused these injuries.

Post-Mortem Examination.

An autopsy at the direction of the City Coroner, Melbourne, was performed on January 10, 1945.

The body was that of a middle-aged, well-nourished male, five feet ten inches in height, and was clad in bathing trunks. Dried superficial abrasions were present on the left shoulder and elbow and on the right wrist. Below and medial to the left nipple was an oval-shaped stab wound (Figure II) half an inch in length and with serrations in its

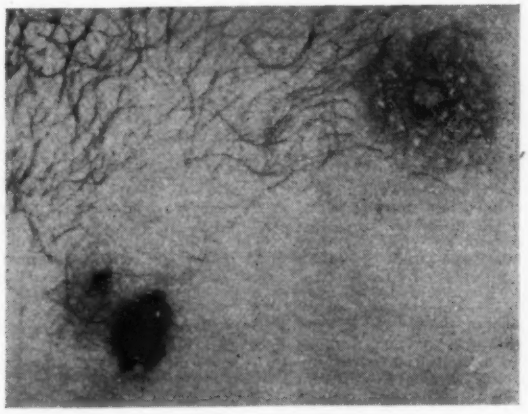

FIGURE II.
Wounds in chest wall (slightly enlarged).

lower border. On the medial side of this wound, at a distance of nearly half an inch, was a tiny rounded puncture wound. The stab wound entered the chest in the fifth left intercostal space, penetrating the left pleural cavity and pericardium through a slightly irregular tear half an inch in length. It then entered the anterior wall of the left ventricle near the apex through a slightly irregular tear three-eighths of an inch in length. The direction was upwards and backwards, and a probe passed through the external wound for a distance of three inches from the surface. The smaller wound did not penetrate beyond the subcutaneous tissue.

The pericardial sac contained fluid blood. The left pleural sac contained a large amount of fluid blood and red clot. Hæmorrhage had occurred in the muscle about the wound in the left ventricle. The cavities of the heart contained a little fluid blood. The coronary orifices and arteries were normal. The valves and aorta were normal. The lungs were red and firm. Moderate œdema was present through the right lung. The left lung was partly collapsed. There was no wound in the tissue of the left lung. The air passages were pale and empty. All other viscera were normal. Death was due to hæmorrhage from a stab wound of the heart.

Comment.

In view of the evidence presented it was concluded that death was due to a stab from a stingray spine. A verdict to this effect was given by the City Coroner, Melbourne, to whom I am indebted for access to the brief of inquest.

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Reviews.

INDIVIDUALITY.

IN "The Biological Basis of Individuality", Professor Leo Loeb has enhanced his reputation by writing a remarkable book.[1] Its scope is so vast, the material in its 714 pages so specialized and intricate, that the reader at times is bewildered. The author draws on a lifetime's experience of experimental biology and human pathology to show us the meaning of individuality. Individuals differ from each other in "individuality differentials". Loeb points out that in actual practice the complete identity of organs, tissues and reactions does not exist. Each individual, however closely related, retains his complex individuality and must be studied from this angle.

The first third of the book is devoted to the transplantation of tissues in the higher vertebrates. It is used as a method for the analysis of the organismal differentials. The surgeon and pathologist will find in these and subsequent chapters much of practical interest. *Inter alia* there is demonstrated the manner in which individuality frustrates attempts at transplantation of organs.

Part II deals with the phylogenetic and autogenetic development of individuality and organismal differentials with the use of the method of transplantation in lower animals. Loeb finds that though differentiation is less rigid than in the higher vertebrates, there are inhibitions based on mechanical and chemical factors.

The author then searches for organismal differentials in the interaction between single cells and discusses infusovians, spermatozoa and ova. He finds that varying degrees of individuality differentials play their part.

Part IV deals with the nature of tumours. In the author's opinion "the organismal differentials in tumors and in normal tissues from which they have developed are identical, or at least very similar, and that the transformation of normal tissues into tumors does not depend upon changes in the genes which determine the organismal differentials, but upon conditions, which are comparable to changes in organ and tissue differentials, although they are not necessarily identical with these". Succeeding sections deal with specific factors in individuality and the role of organismal differentials in the interaction of organ and tissue. Hormones, antigens, blood groups, idiosyncrasy, anaphylaxis and toxins are considered at length.

[1] "The Biological Basis of Individuality", by Leo Loeb; 1945. Illinois: Charles C. Thomas. 9½" × 6¼", pp. 821. Price: $10.50, post paid.

Having dealt with this immense background of factual knowledge, Professor Loeb discusses the problem of evolution. "Evolution is essentially the history of the adaptations between organisms and their environment and between constituent parts within the organism, the non-adapted organisms being eliminated. But there has been an evolution not only in the development of the organisms, their tissues, organs, and their organismal differentials; there has been an evolution also in those processes which lead to the decline of these organisms, such as aging, tendency to disease, and death, all of which are manifestations of the lack of perfect adaptation." The higher organisms have sacrificed plasticity for rigidity of action and interaction.

"While man has thus lost the potentiality to immortal life, he has obtained a greater and richer individuality; he has also gained a life of abstract thought that may help to shape or, if he so desires, to replace the real life and the universe, and he has won a certain degree of consistency and continuity in existence through the persistence of thought and through its transmission to successive generations."

The final section concerns man's further evolution in the social and mental sphere. Human life is a struggle with nature and with other human beings. For this we need material goods, social goods, psychical goods.

Man has largely conquered the first step in his evolution by creating a fixity of body interactions. He must now learn to achieve harmony in an expanded arena of the social, the psychical and the material. Professor Loeb implies that. Mankind travels in a new and chaotic world to which he must adjust himself. This adaptation of the psychical-social environment to the needs of man is in the opinion of the author the most important task which humanity has now to face.

FRACTURES AND ORTHOPÆDICS FOR NURSES AND MASSEUSES.

IT is difficult to write a book to suit the different viewpoints of nurses and masseuses. This Arthur Naylor has attempted in "Fractures and Orthopædic Surgery for Nurses and Masseuses",[1] and the main criticism of the book is that it contains too much detail for nurses and not enough for masseuses. The various orthopædic conditions are well described, but not enough detail is given to aid masseuses in their treatment of them. Occupational therapy and rehabilitation are mentioned only in the introduction.

The book will be of value to nurses who are working in orthopædic wards, and it will help them to take a more intelligent interest in orthopædic procedures and operations. However, the chapter on operative orthopædics is far from complete. Operative technique is glossed over in thirteen lines, and the description of this could have been much improved by the addition of more general details and especially more about the "no touch" technique. Errors which should not have passed the proof-reader also appear in this chapter. Thus in the legend of Figure 49, "Lane bone-bending forceps" should obviously be "Lane bone-holding forceps", while to the end of the legend of Figure 55 the final word "nurse" must be added to make the correct sense.

Fractures are concisely dealt with and the plaster of Paris technique for their treatment is described excellently. The printing and illustrations are of the usual high standard that is expected of the house of Livingstone.

OPHTHALMIC NURSING.

IT is fitting that just as hostilities in Europe and the Pacific has ceased, a fourth edition of Maurice H. Whiting's excellent book on ophthalmic nursing should appear.[2] It has been a standard work for all ophthalmic nurses since its first appearance in 1926. On looking through its 126 pages, one realizes how very little the ophthalmic nurse is expected to know, or rather how elementary is her knowledge when she is qualified. With the great world shortage of ophthalmologists and the increasing complexity of modern ophthalmology, it is clear that in the post-war years the standard of ophthalmic nursing should be raised to a much higher level. This immediately brings forward the thought that the day has arrived when possibly the whole training and status of the general and ophthalmic nurse should be reviewed.

Perhaps it would be wiser in Australia if we put our own house in order first, but that seems to be difficult, as there are only two ophthalmic hospitals in Australia for seven million people, and one of these hospitals also caters for patients with nose and throat conditions. It cannot reasonably be expected that any trained nurse can complete a special course in ophthalmic nursing under twelve months when one takes into consideration that only a small portion of that time is devoted to lectures and study. This is a subject which the Ophthalmological Society of Australia (British Medical Association) could well debate.

The fourth edition of Maurice Whiting's "Ophthalmic Nursing" is warmly recommended to all nurses, both those intending to take special training and also those intending to do private work. There is nothing to surpass it at the moment, and it meets the present requirement of both these sections of the nursing profession.

A TEXT-BOOK OF PATHOLOGY.

IN reviewing the first edition of "Pathology: An Introduction to Medicine and Surgery" by Professor Dible, of the British Post-Graduate Medical School (University of London), and Professor Davie, of the University of Liverpool, we had pleasure in recommending it not only to the student and the general practitioner, but also to the specialist, whether he was surgeon, physician or pathologist. The fact that a new edition has been called for in the short space of six years is in itself an indication of the value of the work.[1]

The number of illustrations has been increased by 21, the text has been reviewed as a whole and rewritten in considerable portions, and, as the authors point out in the new preface, recent knowledge of crush injuries, nephritis, shock, wounds and infections, and the problems arising from blood incompatibility has been included. In addition, there is now a short section on gynæcological pathology (we should have liked to see it larger) and a description of parasites and parasitic diseases, neither of which was included in the first edition.

It should be noted that the subsidiary title is "An Introduction to Medicine and Surgery", which is fully warranted by the inclusion of references to the clinical signs and symptoms associated with, and explainable by, the pathological lesions discussed. The usefulness of this text-book to the general practitioner as well as the student is in this way enhanced. After an account of general pathology, including immunity and infection in general, a series of chapters deal with the special infections, giving an account of the various bacteria and viruses and the lesions that they cause. This is followed by regional pathology and finally by parasitic diseases. As stated in their preface to the first edition, the book does not pretend to be a "repository of information upon all and every sort of pathological condition", but does claim that all the more important pathological processes are fully described. We must say that we do not like the dropping of the capitals in such words as Haversian and Sylvian. On page 722, the reference to Figure 242 should be to 245, and on page 820, that to Figure 238 should be to Figure 368. On page 513, the cells of the lung alveoli are spoken of as endothelial, though usually considered epithelial. These are, however, trifling matters very difficult to eliminate entirely from a work of this size. The coloured illustration of the caecum and ascending colon facing page 656 is an excellent reproduction.

We should like to express our pleasure at seeing this second edition, and can thoroughly recommend it to the student and the practitioner.

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[1] "Fractures and Orthopædic Surgery for Nurses and Masseuses", by Arthur Naylor, Ch.M., M.B., M.Sc., F.R.C.S. (England), F.R.C.S. (Edinburgh), with a foreword by Ernest Finch, M.D., M.S. (London), F.R.C.S. (England); 1945. Edinburgh: E. and S. Livingstone Limited. 8½" × 5", pp. 300, with 243 illustrations. Price: 16s. net.

[2] "Ophthalmic Nursing", by Maurice H. Whiting, O.B.E., M.A., M.B., B.Ch. (Cantab.), F.R.C.S., Fourth Edition; 1945. London: J. and A. Churchill Limited. 7½" × 5", pp. 143, with 56 illustrations. Price: 6s. 6d.

[1] "Pathology: An Introduction to Medicine and Surgery", by J. Henry Dible, M.B. (Glas.), F.R.C.P. (Lond.), and Thomas B. Davie, B.A. (Cape), M.D. (Liverp.), F.R.C.P. (Lond.); Second Edition; 1945. London: J. and A. Churchill Limited. 9½" × 6½", pp. 956, with 395 illustrations, including 8 plates in colour. Price: 45s.

The Medical Journal of Australia

SATURDAY, DECEMBER 22, 1945.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE PROBLEM OF DENTAL HEALTH IN AUSTRALIA.

A few weeks ago a good deal of space was devoted in these columns to a discussion of the eighth interim report of the Parliamentary Joint Committee on Social Security. The report covered such a wide range of subjects that discussion of at least two of them was allowed to stand over for another occasion. One of these subjects had to do with the provision of a dental health service for the Commonwealth. Too many people in the community think of dental health, if they think of it at all, as something apart, a kind of refinement and altogether unessential. Even medical practitioners who know that preventive dentistry is as much an ideal to be sought as preventive medicine, do not all appreciate the magnitude of the problem and with it the enormous difficulty of finding a solution. When the Parliamentary Joint Committee on Social Security was inquiring into the provision of a comprehensive health service for Australia, evidence was naturally taken from individual dental practitioners and from the Australian Dental Association. A section of the eighth interim report is devoted to a discussion of this evidence. We propose first of all to give a short account of the main features of the problem and then to indicate the attitude of the dental profession to the whole question.

The first statement regarding the problem is arresting and holds a challenge for the medical as well as for the dental profession—for the medical profession particularly in regard to children, for its members have access to homes in the community and come into contact in one way or another with the majority of young children. The statement is that dental disorders affect 95% of the population, but that 75% of the members of the community are unconcerned with, if not ignorant of, the "clearly defined importance of prevention". In case any should object that in the departmental medical examination of school children inspection and a certain amount of treatment are carried out, it must be observed that in every place the work done is only a fraction of what should be done and of what could be done if provision was made for adequate staffing and equipment. In a memorandum of the evidence given by the Australian Dental Association, published separately by that body, we are reminded that the ravages of decay are seen in the mouths of most children who do not receive regular treatment, and it is suggested that not more than 15% of children obtain this care. The percentage of people in the community who receive no dental attention or only that required for the relief of pain, is 75. This means that the existing dental personnel of the Commonwealth will have to be at least trebled if any demand for a national standard of dental fitness is to be met. The Australian Dental Association is pessimistic about adults—"there is no possibility of combating or overtaking the widespread disease in every dentally crippled adult, however tempting it might be to contemplate such a prospect theoretically". This is easy to understand both from the point of view of dental personnel and also because habits of personal hygiene are acquired in childhood and "an adult seldom changes his habits except for the worse". The magnitude of the treatment problem is illustrated by the following facts. There are in Australia 982,123 children between the ages of two and ten years, and it is assumed that of these 864,286 are in need of treatment. The average number of fillings required is ten. Thus at least 8,500,000 fillings are needed by children of this group. Even if these fillings could be made, the recurrence of dental caries would be overwhelming unless subsequent regular treatment was carried out. If any national dental service is established, the Australian Dental Association recommends that a start should be made with a small age group, two to four years inclusive. In this group the incidence of caries is less than in older groups, and it is thought that once a satisfactory start has been made, it might be possible to add another age group every year until all children of school age had been included. In the two to four years age group there are 345,150 children. If an allowance is made for the 15% of children who are already receiving treatment, approximately 300,000 children require initial treatment. This would need the whole-time attention of 600 dental surgeons for one year. From this estimate a natural inquiry will be into the number of dental surgeons in Australia. On present information available to the Central Dental Manpower Advisory Committee there is one dentist to every 2,171 persons in the Commonwealth. The number of patients of whom one dentist can take care is difficult to determine. In the armed forces it has been found that 1,000 are too many for one dental officer. From calculations made at the Dental Hospital of Sydney it is thought that possibly 500 patients requiring initial treatment would represent a reasonable annual average per dentist and that for subsequent maintenance purposes double this number could be treated.

The urgent need for the future, if adequate dental attention is to be given to the community, apart altogether from the possible establishment of a national dental service, is an increase in the number of persons permitted to practise dentistry. The Australian Dental Association suggested in its evidence that expansion of personnel might be derived from two sources, by an increase in (*a*) the number of qualified dental surgeons, (*b*) the number of assistants who would relieve dental surgeons of work which did not require the exercise of full professional knowledge and skill. As the establishment of a national

dental service is the main object of the Parliamentary Joint Committee's inquiries, it is necessary to emphasize the Australian Dental Association's reasonable contention that the planning of such a service should be preceded by the passing of a federal dental act which would provide for the uniform Commonwealth registration of dentists. At the present time university degrees in dentistry are granted by the Universities of Sydney, Melbourne, Adelaide and Queensland. Tasmania has no faculty of dentistry, and in Western Australia a diploma is granted by the Dental Board of Western Australia to persons who have completed a prescribed course at the Western Australian College of Dental Science. The college is entirely separate from the University of Western Australia—a state of affairs related apparently to the fact that no fees are charged at the University of Western Australia. This same kind of difficulty, incidentally, would probably arise if attempts were made to establish a medical school in Western Australia. If a solution was urgently sought and the University of Western Australia could not modify its arrangements to absorb the College of Dental Science, the alternative would be the conferring of university status on the college by special act of parliament. When this was achieved the question of uniform registration would still remain insoluble for constitutional reasons. If the States wished to achieve uniformity of registration under a Commonwealth act, they would have to be willing to surrender their sovereign rights in the matter to the Commonwealth. It may not be out of place to remark that ever since this journal came into being it has not ceased to urge the introduction of uniform medical registration. This, however, is taking us away from our discussion on an accession of personnel to the ranks of the dental profession. No doubt more students will enter the faculties of dentistry at the several Australian universities now that hostilities have ceased, but they will soon be overcrowded, if they are not too full already, and a position similar to that existing in the faculties of medicine will arise. The Australian Dental Association refers to the claims of technicians as fit and proper persons to receive a modified form of training with examination concession and special facilities for becoming registered dental surgeons without university graduation. The suggestion is examined carefully and (we think rightly) rejected. The proposal is "an expedient for obtaining easy access to a profession that bears an important responsibility to the health of the community, without facing the discipline of proper scientific training so necessary to render a skilful and honest professional service". Another statement which carries conviction is that "as the care of children's teeth is the logical basis for any national service, it cannot be convincingly accepted that preventive and conservative treatment of the juvenile has any appeal to minds developed entirely on lines of the artificial replacement of teeth"—what is commonly known as "blood and vulcanite". The Australian Dental Association in its evidence referred with approval to the work of school dental nurses in New Zealand who undertake the treatment and extraction of children's teeth and give instruction in dental hygiene. These nurses undergo a period of two years' intensive training in this restricted field at a college especially established for that purpose. It was pointed out that while the dental profession in Australia viewed the project with mixed feelings, varying from enthusiasm to misgivings, it had unquestionably succeeded in New Zealand. In America "dental hygienists" carry out prophylactic work, and it has recently been suggested that their training should be enlarged to prepare them to carry out duties similar to those of the New Zealand nurses. It is to be noted that although the Australian Dental Association urged this type of dental worker before the Parliamentary Joint Committee on Social Security, the Victorian Branch has since withdrawn its support of the recommendation. (This is stated in the preface of the Australian Dental Association's memorandum.) Opinions in the medical profession will vary on such a proposal. Probably the majority of medical practitioners will be very hesitant to approve of the idea. The Australian Dental Association refers to children's dentistry as a very restricted field. Apparently the suggestion has not been made that the two years' intensive training for admission as a "dental hygienist" should take place at a university, the time if necessary being expanded somewhat to include an adequate scientific background, and that a diploma might be issued at the end of that time, enabling the candidate to practise as a children's dentist. If such an expedient were adopted the granting of a degree in dental surgery to a dental hygienist would be contingent on a further period of undergraduate study at a university and the passing of the requisite examination. It would be interesting to know how a suggestion of this kind would be received by members of the dental profession.

The remaining aspect is the attitude of the dental profession towards the creation of a national dental service. The general attitude is described in the following quotation:

> The members of the profession approach the question of a national dental health scheme with varying degrees of scepticism, with an attitude of full co-operative support for a plan that will advance the standards of dental fitness, particularly among children, and with strongly pronounced convictions against the introduction of any scheme that might jeopardise the present standard of dental science or abolish the right of private practice. The public must not be deprived of the right to select their own dental surgeon and obtain the private treatment they have sought and secured for so many years.

The Australian Dental Association was emphatic that if a dental health service was established, it should be under the administrative control of a commission constituted as follows: (*a*) A chairman and two other dental members selected from a small panel nominated by the dental profession (one should have had administrative experience of dental hospitals, clinics or organizations); (*b*) the Commonwealth Director-General of Health; (*c*) a lay representative with special qualifications and experience in finance. Finally the following three paragraphs may be quoted:

> The association is strongly opposed to any plan that implies nationalisation of the profession. Regimental control and mass production lead to deterioration in standards of efficiency, and the loss of incentive ultimately results in stasis of individual efforts.
>
> The impossibility of implementing any plan rests upon the fact that there are not sufficient qualified dentists in Australia without supplementing dental personnel. Dental treatment from childhood to senescence for all members of the community would require the present number of dental surgeons, approximately 3,330, to be trebled.
>
> Interference with the existing system of private practice, wherein the individual members of the community have the right to select the practitioner of their choice, is contrary to democratic principle.

The Parliamentary Joint Committee on Social Security comes to three important conclusions and with a statement

of these we must rest content at present. The first is that no comprehensive health service would be complete without adequate control of dental diseases, and that the inauguration of a national dental service should form part of the complete plan. The second is that such a service covering the problems of research, prevention, treatment and personnel should be planned by representatives of the dental profession and the public, and be administered by the profession itself. The third conclusion is that uniform registration of dentists throughout Australia will be a necessary step before such a service can be satisfactorily established.

Current Comment.

WHEATMEAL, OATMEAL AND PHYTIC ACID.

ON March 11, 1942, the British Minister of Food announced that after April 16 of that year the national loaf was to be made with an 85% extraction flour. This step was not dictated by immediate considerations of the nutritive quality of the new bread, which the lay Press so frequently and so erroneously referred to as "whole-meal bread", but arose from shipping difficulties. The view was that if people ate bread containing more of the wheat grain less grain would have to be imported and more space would be available for military material. The suggestion to make flour with over 90% of the wheat grain was rejected for a number of reasons, one being the reduced absorption in the digestive canal of the consumer, also the poorer keeping qualities and the needs of animal husbandry. Two opposing views soon became manifest in Britain. On the one hand there were those (and they are still vocal) who advocated a true whole-meal bread. Their main argument was the unassailable fact that the pericarp of the wheat grain is rich in vitamins, particularly those of the *B* group, and also in minerals, notably phosphate and iron, whereas the endosperm is poorly supplied with these. On the other hand, the general trend of popular taste was in favour of white bread, which is attractive in flavour and appearance, has good keeping qualities, has a higher calorie value, and gives a smaller unabsorbed residue. Government regulation favoured popular sentiment, and the extraction of national flour was reduced to 82·5%, and then on January 1, 1945, to 80%.

A completely new aspect was given to the debate, often conducted with needless acerbity of expression, by the discovery of the action of phytic acid, which hinders the absorption of calcium and leads to an adverse calcium balance. White flour of 70% extraction was found by Widdowson to contain 51 milligrammes of phytic acid per 100 grammes, national wheat meal of 85% extraction 127 milligrammes, and wheat meal of 92% extraction 214 milligrammes.[1] Laboratory analyses might display a richness of minerals, including phosphates, in the wheat pericarp, but the formation of phytates reduced the amounts absorbed well below those chemically demonstrable. As is well known, *creta præparata* was added to national flour to counteract the phytic acid arising from the pericarp. The injurious effects of flour of over 90% extraction have been demonstrated on a large scale in Dublin.[2] Irishmen in rural districts make potato and oatmeal the basis of their diet, the phytic acid of the oat grain being neutralized, so the theory went, by the high calcium content of the milk invariably taken with porridge; but Dublin folk are bread-eaters and exigencies of shipping led to a high extraction flour. The untoward result has been a disquieting increase in rickets.

A recent contribution to the phytic acid problem has emanated from the Department of Physiology and the Rowell Institute in Aberdeen.[1] Four healthy adults were given diets with preponderating oatmeal content and the lime and iron metabolism was examined quantitatively. The quite unexpected result was obtained that the phytates were found to be almost completely digested. This means that not only was calcium absorption not hindered, but that the phosphate of the phytic acid was given to the body in assimilable form. When calcium was administered in excess of the body's needs, there was some reduction in the digestion of the phytates. McCance and Widdowson found that in adults taking wheat flour containing phytic acid an average of 46% of the ingested phytic acid was excreted unchanged.[2] The authors can give no reason for the different digestion of wheat and oat phytates. One possibility suggests itself: the subjects of the experiments were residents of northern Scotland and may have been accustomed to eating oatmeal in liberal quantities. As is well known, the Scot, whether through early initiation or through a process of natural selection, can thrive on an oatmeal preponderance which would make his neighbour south of the Tweed very unhappy. Anyhow, here is another feather in the cap of oatmeal porridge.

[1] *Nature*, Volume CXLVIII, 1941, page 219.
[2] *British Medical Bulletin*, Volume II, 1944, page 221.

ALLERGY AND THE CENTRAL NERVOUS SYSTEM.

THAT the central nervous system can be involved in allergic reactions has been known for some years. Attacks of narcolepsy, insomnia, persistent yawning, even hemiplegia and transitory aphasia, also anxiety states and fatigue have been ascribed to specific allergens. In 1941 Crowe described a case of a sixteen-year-old boy, allergic to rice, peas and spinach, displaying in the reaction nausea, vomiting, spasmodic contractions of head and limbs, narcolepsy and œdema of the face and one orbit, the attacks lasting ten to twelve hours and recurring once to three times a month.[3] This author announced his belief that cerebral allergy could produce changes of personality and mental deterioration. The description of a case of considerable interest has recently been published by the Faculty of Medicine in Montevideo, Uruguay.[4] Though no new facts of major importance have been added to what is already known, the pathological picture was so clearly outlined as to leave no dubiety as to the cause. A boy of fourteen suffered recurrent attacks of violent hemicrania with visual disturbance and hemianæsthesia. When the hemianæsthesia was right-sided there was aphasia, when left-sided no aphasia. Cerebral œdema of allergic origin was undoubtedly the provocative factor. One very remarkable aspect of the case was the number of substances to which the boy was allergically responsive. Amongst vegetable foodstuffs giving the reaction were wheat, yielding a particularly violent skin reaction, water cress, broccoli, onion, cauliflower, endive, spinach, leek, red beet, hazel nut, rye, chick pea, coffee, strawberry, lemon and melon; in the list of animal foods were pork, beef, lamb, tunny, cod, both fresh and salted, and sardine; inhalations which excited responses were those from feathers of duck and partridge and the fur of the cat. To give the boy sufficient nourishment and yet avoid the allergens was a difficult task, particularly as the young patient had been accustomed to eat ten to twelve bread rolls (*panes*) in the day, and wheat, as stated, headed the list for severity of reaction. Desensitization procedures improved the condition considerably, for after treatment four months elapsed without any attack. That angioneurotic œdema may occur in other structures than the cutaneous and superficial is a possibility which the clinician must always bear in mind, for many transient pathological states may find in this their explanation.

[1] Cruickshank, Duckworth, Kosterlitz and Warnock: "The Digestibility of the Phytate P of Oatmeal in Adult Men", *Journal of Physiology*, June, 1945, page 41.
[2] *The Biochemical Journal*, Volume XXIX, 1935, page 2694.
[3] *Journal of Allergy*, 1941, page 172.
[4] Alejandro H. Schroeder: *"Jaqueca con Hemianestesia y Afasia a Repetición de Origen Alérgico"*, *Anales de la Facultad de Medicina de Montevideo*, Volume XXX, 1945, page 277.

Abstracts from Medical Literature.

THERAPEUTICS.

Thiouracil.

S. L. CARGILL AND M. F. LESSES (*The Journal of the American Medical Association*, April 7, 1945) have discussed toxic reactions to thiouracil. Thiourea proved to have so many toxic effects that its use was discontinued and thiouracil was substituted in its place in the treatment of toxic goitre. Thiouracil also has serious and unpredictable toxic effects. Agranulocytosis, drug fever, swelling of salivary glands, dermatitis and lymphadenopathy have been recorded. In the authors' series of 43 patients suffering from toxic goitre and treated with doses of thiouracil ranging from 0·1 to 1·0 gramme daily, there occurred one fatal case of agranulocytosis, one non-fatal case of granulopenia, two cases of jaundice, two of drug fever and two of swelling of submaxillary glands. The patient who died of agranulocytosis was treated with 0·6 gramme of thiouracil daily for the first seventeen days, 0·4 gramme daily for the next six days and 0·2 gramme every day thereafter. The basal metabolism fell from +30% to −2% and the patient left hospital better in every way. The basal metabolism fell to −18% after three months; the patient was irritable, sluggish and puffy about the face. Thiouracil was omitted because of myxœdema. The leucocyte count was between 6,000 and 10,000 per cubic millimetre during this early period. Thiouracil was remitted for six weeks and resumed in doses of 0·2 gramme daily, but after eleven weeks was remitted again on account of myxœdema. After seven weeks thyreotoxic symptoms recurred and thiouracil was resumed in doses of 0·2 gramme daily for one month. At this stage a sore mouth, fever and prostration developed, and the number of leucocytes fell to 1,000 per cubic millimetre with no polymorphonuclear cells. Three transfusions and sodium sulphadiazine and sulphamerazine were given, 270 mils of "Pentnucleotide" were given intramuscularly, and liver extract and 150,000 units of penicillin were administered. The patient died on the sixth day. The second case of agranulocytosis developed after the patient had received 0·8 gramme for seven days, 0·6 gramme for eighteen days and 0·4 gramme for three days. In the fourth week of treatment agranulocytosis developed, neutrophile cells falling first to 30% and then to 7%. Seven hundred and fifty mils of blood and 40% "Pentnucleotide" given daily for two days were followed by rapid recovery. Pruritus and urticaria appeared temporarily. Two patients developed severe jaundice after three weeks' treatment with thiouracil in doses of 0·6 to 1·0 gramme daily. There were clay-coloured stools and dark urine, no pruritus, abdominal pain or nausea; the jaundice lasted 100 days in one case, 26 in the other. Two cases of drug fever occurred, one after 1·0 gramme daily for eleven days and 0·6 gramme daily for seven days, the other after 1·0 gramme for nine days. Fever up to 102° F. occurred for two to three days, and in one case readministration of the drug was followed by recurrence of fever. The temperature fell when the drug was omitted. Submaxillary gland swelling was noted in two cases after one month on 0·4 gramme daily; the drug was continued and the glands returned to normal in seven to fourteen days. Toxic symptoms have been observed after small doses for a few days, and at present it is impossible to foretell in which case they will appear. The authors emphasize the dangers of thiouracil and insist on careful control during its administration.

Curare and Anæsthesia.

H. R. GRIFFITH (*The Lancet*, July 21, 1945) discusses the value of curare as a muscle relaxant in patients under anæsthesia induced by cyclopropane, ether and other agents. He has chiefly used "Intocostrin", and a new preparation, "D-tubocurarine", neither of which is irritating to the subcutaneous tissues. He administers curare only when increased muscular relaxation is needed; this may be done at any time during the operation, and the dosage should be adequate (60 to 100 milligrammes for an adult under cyclopropane anæsthesia). The dosage may be repeated, but this is not necessary in every case of anæsthesia for abdominal surgical measures. The author states that he has used curare 500 times, and uses it on the slightest provocation; yet he has needed to use it in only 38% of abdominal operations. "Curare should not be made the excuse for a poor anæsthetic sloppily administered." He prefers cyclopropane as an anæsthetic agent, and has found that cyclopropane and curare make an ideal combination, producing relaxation equal to that obtained in spinal anæsthesia. Curare may also be used in anæsthesia induced by ether, or by ether combined with cyclopropane, with ethylene or with nitrous oxide; but in these circumstances the dosage of curare should be greatly reduced (an average of 20 to 40 milligrammes for an adult). The author urges greater appreciation of ethylene, which he considers a particularly safe anæsthetic agent in "poor risk" cases, and doubly efficient when combined with curare. Curare may also be used to reinforce the action of "Pentothal", and may be given to patients in whom the relaxation of spinal anæsthesia is wearing off too soon, provided hypnotics or a "sleeping dose" of general anæsthetic is given. Curare may be given intramuscularly, but the author prefers to give it intravenously. The modern standardized curare extracts are rapidly broken down in the human body. When curare is given intravenously, it acts within thirty seconds, maintains its maximum effect for about five or ten minutes, and then gradually disappears until its effect is all gone in about twenty minutes. This rapidity of elimination is its greatest safety factor, and recent reports have shown that very large doses may be given without irreversible damage to the patient; but very large doses are not advocated. Occasionally during anæsthesia an average dose of curare may cause a patient's respiration to become shallow or to cease; this is due to the paralysing effect of the drug on the accessory muscles of respiration and finally on the diaphragm. The only treatment necessary is artificial ventilation of the lungs by manual compression of the breathng bag during the few minutes before the depressing effect passes off; the author has never seen even temporary damage to the patient result.

Bacterial Endocarditis.

M. MEADS, H. W. HARRIS AND M. FINLAND (*The New England Journal of Medicine*, April 26, 1944) discuss the treatment of subacute bacterial endocarditis with penicillin. In nine infections caused by *Streptococcus viridans* and seven proved or probable cases caused by other organisms the patients were treated with sodium penicillin at the Boston City Hospital during 1944. Several blood cultures were obtained in each case before treatment was begun. The penicillin was usually given by repeated intramuscular injections and rarely by other methods; 25,000 units were given every two hours to begin with. The organisms were tested for their sensitivity to penicillin by the method of Rammelkamp and Maxon. Treatment was continued for twelve to nineteen days. Heparin was used in three cases, but was stopped in two owing to untoward symptoms. Three patients died out of nine treated. The dose of penicillin was varied in some cases according to circumstances; occasionally when the response seemed unsatisfactory or when a recurrence of symptoms took place, 15,000 units were injected every hour. In two cases bacteriæmia recurred and responded to another course of penicillin. Sulphonamides were used in this series of cases only as supplementary treatment and for coexisting infections. The cases of acute bacterial endocarditis were suspected in persistent bacteriæmia when embolic phenomena or cardiac murmurs appeared and persisted. Two patients suffering from pneumococcal septicæmia recovered and one died after one to three million units of penicillin. One patient suffering from gonococcal and one from staphylococcal endocarditis died. Penicillin is the most effective agent in treatment of subacute and acute bacterial endocarditis.

Aluminium Hydroxide in the Treatment of Peptic Ulcer.

E. N. COLLINS (*The Journal of the American Medical Association*, April 7, 1945) describes the treatment of peptic ulcer with aluminium hydroxide. He states that this method, which has been used in the treatment of 3,000 patients at the Cleveland Clinic in the past eight years, is as satisfactory as treatment with absorbable antacids. He considers aluminium hydroxide gel superior to calcium carbonate, first because there is no tendency to formation of renal calculi and secondly because the antacid effect is maintained over a longer time. In the first month, milk feedings are given every two hours, and aluminium hydroxide gel is given every two hours at alternate hours with the feedings. The dose is never less than two fluid drachms. The bed-time dose is never less than one fluid ounce. In the second and third months, an interval feeding of milk is given at least once between meals. The antacid is given six times a day—one hour after meals, one hour after interval feedings and at bed-time. During the remainder of the first year, one interval feeding is given between meals. Aluminium hydroxide gel is given four times a day—one hour after each meal and at bed-time. The author has found liquid alumina gel preparations more effective than solid prepara-

tons. The treatment does not completely neutralize gastric acidity, but keeps the level of free acid low enough to prevent the activation of pepsinogen. Peptic digestion does not occur if the gastric contents are maintained at a pH of 3·5 or higher. In addition, pepsin has been found to be precipitated and inactivated by the alumina gels.

The Treatment of Obesity by Appetite Control: The Use of Autonomic Substances and their Synergists.

LOUIS PELNER (*Annals of Internal Medicine*, February, 1945) considers that the most common and important type of obesity is due to over-indulgence in food. He stresses the relative rarity of glandular causes, and thinks that Fröhlich's syndrome is diagnosed more often than it actually exists. True Fröhlich's disease, Cushing's disease and hypothyreoidism cause a certain degree of adiposity. Low calorific diet is of the greatest importance, and if adhered to, no other treatment is required. Psychotherapy is often necessary as well, and the clinician should explain to girls the value of an attractive figure, and to older patients the possible damage to the cardiovascular system. A diet of 1,000 to 1,500 Calories a day is prescribed with a low fat and low carbohydrate content; salt and water intake should be restricted. Adherence to a diet with a low carbohydrate content alone will often cause intense weakness. For this reason it is necessary to employ means that will curtail the appetite and at the same time give the patient a feeling of well-being that will encourage him to continue the dietary treatment. Benzedrine, atropine and if necessary thyreoid extract have been included in the capsule. Benzedrine 5 milligrammes increasing to 10 milligrammes, atropine 1/750 grain increasing to 1/250 grain, and thyreoid extract a half to one grain were given three times a day one hour before meals. Owing to water retention, women do not usually lose weight around menstrual period time. During this time aminophyllin was given in doses of three grains three times a day, and often produced a satisfactory loss of weight. After several months, a point was reached at which the patient became refractory to both benzedrine and atropine. Recommencement of therapy with smaller dosage, after a rest of two weeks, was again effective. Iron and vitamin supplements are given. The author discusses the mode of action of benzedrine and atropine, and also the value of a gel at the end of treatment. Exercise is of benefit in the management of the obese patient.

NEUROLOGY AND PSYCHIATRY.

Psychosomatic Aspects of Stuttering.

STUTTERING, according to Bernard C. Meyer (*The Journal of Nervous and Mental Disease*, February, 1945), though occurring ten times more frequently in the families of stutterers, does not appear to follow any simple Mendelian hereditary pattern. Neurological examination of 116 stutterers failed to reveal any significant organic disease or constitutional organic inferiority. The most frequently observed symptoms were those of chronic anxiety. Psychiatric examination revealed a number of schizoid individuals: in many there was a history of enuresis, nail-biting, nightmares and other features of morbid emotional tension. The author regards stuttering as but a single manifestation of a more general neurotic problem—the resultant of the conscious will to express itself and an unconscious inhibition of speech. This latter appears to serve as a defence against anxiety.

Morbidity and Mortality of Patients with Psychosis due to Cerebral Arteriosclerosis.

O. J. POLLAK (*The Journal of Nervous and Mental Disease*, July, 1945) has studied the morbidity and mortality of patients with psychosis due to cerebral arteriosclerosis. His study covers two decades and includes 2,000 necropsies, among which were 476 in which the subjects had been diagnosed as suffering from psychosis with cerebral arteriosclerosis. Only in one-fourth of these cases was death due primarily to arteriosclerosis. Average age at death was seventy-one years, alike for both sexes; but males whose death resulted directly from cerebral arteriosclerosis only reached an average age of sixty-four. Other mental patients showed a much lower mortality from arteriosclerotic processes, and, except for the senile cases, a lower average age at death.

Functional Symptoms as First Evidence of Pancreatic Disease.

IN presenting the histories of three patients displaying functional symptoms of pancreatic disease, N. K. Rickles (*The Journal of Nervous and Mental Disease*, June, 1945) states his belief that in many cases pathological change involving the pancreas has been overlooked owing to the masking of the true pathological condition by symptoms of profound depression and anxiety. In the first case, a man of fifty-seven complained of anxiety, depression and abdominal pain, and in the absence of clinical findings was diagnosed as suffering from involutional melancholia. Gastro-intestinal investigation and chemical examination of the blood revealed no abnormality. The subjective symptoms became more severe, and exploratory laparotomy disclosed an inoperable carcinoma of the pancreas. Similar histories were obtained in two other cases. The author concludes that the clinician should be ever on the alert for pancreatic disease when deep-seated epigastric pain is associated with mental symptoms and an absence of clinical signs.

Pathology of the Liver in Extrapyramidal Disease.

FINDING the literature relating to the structural and functional changes of the liver associated with extrapyramidal disease contradictory, except in the case of hepatolenticular degeneration, G. Heilbrunn, O. Felsenfeld and P. Szanto (*The Journal of Nervous and Mental Disease*, July, 1945) investigated cases of Huntington's chorea, post-encephalitic Parkinsonism and Parkinson's disease. Using delicate liver function tests, such as the colloidal gold test of the blood serum, the determination of the prothrombin time, Weltman's coagulation band and the Lange-Heuer flocculation test, and investigating partial sectors of the protein metabolism of the liver, the authors obtained positive readings in 85% of the 54 cases of extrapyramidal disease studied. The highest percentage of positive readings was in Huntington's chorea.

General Paresis in Senility.

GENERAL PARESIS is considered by Silvand Arieti (*The American Journal of Psychiatry*, March, 1945) as occurring in senility when its initial manifestations appear at or after the age of sixty. After reviewing the literature on this subject, he introduces in detail six new cases. The incubation period in these senile types may be from forty to fifty years. Diagnosis is rendered more difficult if serological examination of the blood reveals no abnormality. Cases may be divided into two groups: (*a*) those characterized by acute cerebral inflammatory changes with the absence of arteriosclerotic symptoms; (*b*) those characterized by milder inflammatory changes and the almost constant presence of arteriosclerotic lesions. The second group is more common than the first. A study was made of the relationship between the paretic process, cerebral arteriosclerosis and senility, which seemed to suggest that the presence of cerebral syphilis inhibited the neuropathological changes of senility, but might accelerate the arteriosclerotic changes.

The Homosexual as a Personality Type.

HERBERT GREENSPAN AND JOHN D. CAMPBELL (*The American Journal of Psychiatry*, March, 1945) consider that the true homosexual exists as a distinct personality type. They believe that its origin is always constitutional and never environmental or acquired. They contend that physical and psychic manifestations of effeminacy are characteristic of the invert. Observations, they claim, drawn from androgen-œstrogen urine levels in homosexuals tend to enhance the conviction that homosexuality is constitutional in nature. The authors believe that, though homosexual activities occur among psychopaths, psychotics, schizoid personalities and the feeble-minded, it is wrong to infer that such occurrences indicate the true homosexual personality. Clinical differentiation is made from the normal personality, the mental defect, and the psychotic personality, the psychoneurotic and the schizoid. They believe that true homosexuality is not an acquired vice but a biological anomaly.

The Electrical Diagnosis of Peripheral Nerve Injury.

ANTHONY E. RITCHIE (*Brain*, Volume LXVII, Part 4, 1944) describes a technique for the routine determination of strength-duration curves of patients suffering from peripheral nerve injury. The examination affords a quantitative expression of the accepted galvanic-faradic test for the so-called "reaction of degeneration". By the utilization of this technique characteristic quantitative differences may be observed in the excitability of normal and denervated muscle. The author states that improvement in excitability is noticeable a short time before the earliest signs of recovery can be detected by clinical means. This is of special prognostic value.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Section of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association, in conjunction with the Mental Hospitals Department of New South Wales, was held at the Mental Hospital, Rydalmere, on June 21, 1945. The meeting took the form of a series of clinical demonstrations arranged by DR. S. J. MINOGUE, the Medical Superintendent of the hospital.

Attempted Suicide.

The first patient shown was a male, aged thirty-six years, who had been admitted to the hospital on February 19, 1945. He was too non-cooperative for his intelligence quotient to be estimated. There was nothing of importance in the patient's family history. At the age of four years he had suffered from a cerebral abscess and had been mentally retarded since then. He was reared by his sisters, and became an invalid pensioner at the age of sixteen years. He had apparently given no trouble until a month before his admission to hospital, when he talked of getting married. He was taken by a girl to the manpower authorities, and obtained a job at a glass works, which he kept for three days. After that he went about telling people that he could make a baby in a month. His married sister, with whom he was living, then grew afraid that he would become dangerous to her two small daughters, and had him certified insane. The patient himself said that his brother-in-law, a wharf labourer, had stolen rationed goods in his possession, and wanted him to go round selling them. Because he refused to do this, he was sent to the hospital.

Since his admission to hospital the patient had been restless and troublesome, and had not settled down. Three weeks prior to the meeting he had attempted to strangle himself. One week later he broke from the ward and waded into the river. He became terrified, and waded around the foreshores and came out again. Three days prior to the meeting he attempted to swallow a large rabbit bone. These suicidal attempts were considered to be in the nature of hysterical episodes.

Epilepsy.

The second patient was a male, aged twenty-five years, an epileptic, who had been admitted to the hospital on January 5, 1945. His intelligence quotient was 75%. The patient was an illegitimate child, and had been adopted at the age of six months. He had suffered from convulsive seizures since the age of six months. He was backward at school, and had been an invalid pensioner since the age of sixteen years. His hobby was repairing watches; but his degree of efficiency was unknown. He had lived at home with his adopted mother. The patient admitted masturbation, and consorted with prostitutes about once a month. He had never given any trouble until December 29, 1944, when he wrote to the father of a girl, aged ten years, stating that he intended to kidnap and criminally assault her. He signed his name to the note. The patient admitted that he wrote the note, but said that it was all a joke. He had no idea of morality and wanted the loan of his sister-in-law for nine months. His mother had asked for his release from the hospital, and a police report was obtained. The evidence was that he had never been known to be a menace to girls; but the father of the girl about whom the patient had written the note objected to his release, on the grounds that he would be a menace to the girls in the neighbourhood. On April 22, 1945, the patient escaped from his mother in the grounds, and was returned on April 24. During this time he had visited his prostitute.

Exhibitionism.

The third patient shown was a male, aged nineteen years, who had been admitted to the hospital on July 14, 1944; his intelligence quotient was 64%. Nothing of importance was learnt from the family history. The patient had always been backward at school; although he said that he had been in first year, his general knowledge was extremely poor. On leaving school he had worked as a labourer in a factory, and had apparently given no trouble until he was aged seventeen years, when he exposed himself to two women. The charge against him at that time was withdrawn. He admitted a history of masturbation prior to that episode. On July 5, 1944, he was charged with stealing from a backyard a collection of female clothing; he walked into a lane, undressed, put on part of the stolen clothing and destroyed the rest. He then put on his own clothing. He admitted the crime, but could give no valid reason for it. Since his admission to hospital he had been a model patient and an excellent ward worker. He had shown no signs of sexual perversion.

Alcoholism and Sex Delinquency.

Another patient shown was a female, aged twenty years, a full-blooded aboriginal, who had been admitted to hospital on May 21, 1943; her intelligence quotient was 44%. She had a history of alcoholism and sex delinquency; she had had an illegitimate child, and negected it. She was a confirmed alcoholic and promiscuous with men. She had always been careless and untidy in her dress and neglectful of her personal hygiene. Since her admission to hospital she had been well-behaved. She worked well as a rule, but at times became very lazy. She had escaped once since her admission to hospital.

The comment was made that the patient was an aboriginal, out of her element. Living constantly in touch with civilization, she had copied the vices but not the virtues of the white people. She would probably do well on a mission station amongst her own people far from populated areas, and negotiations with the Aborigines Welfare Board were being undertaken so that she could be placed in a suitable environment.

Moral Imbecility.

Another patient shown was a female, aged seventeen years, who had been admitted to hospital on March 14, 1945; she had an intelligence quotient of 53%. The girl was the eldest of a family of six; she had reached third class at school, but was unable to learn. She had been promiscuous since the age of thirteen years. If she was corrected at home, she used to run away. Eventually her mother took action against her as an uncontrollable child, and the patient had been in and out of an industrial school for the past four years. She was certified insane because she was one of the ringleaders in a series of riots at the industrial school. In hospital she worked well, but had outbursts of uncontrollable temper, during which she smashed furniture, used filthy language and attacked staff and patients. The comment was made that she was a moral imbecile and would never reform.

Moral Delinquency.

A further patient was a female, aged eighteen years, who was said to have been always backward at school, but to have much worldly wisdom; her intelligence quotient was 56%. The patient became a moral delinquent at the age of thirteen years, when she was found in a residential with her mother and two soldiers. Her father was overseas on service with the Australian Imperial Force, and she and her mother had been associating with men in many residentials. The patient was committed to a convent, but absconded twice, and was then sent to a girls' industrial home. She absconded from there, and when taken back became insolent and troublesome. She was then certified insane and sent to the Mental Hospital, Kenmore, where she became a model patient. After some months she was allowed to go on leave to her father. She apparently remained well for ten months, when she was found, naked and drunk, in a residential with another girl and two soldiers. She was returned to the hospital on May 18, 1942. After some months she was again allowed out on leave to her father, and remained out for eight months until October, 1944. On her return she accused her father of incest; but the evidence against him was so flimsy and the patient was so unreliable that the police would take no action. In January, 1945, she escaped from the hospital, but was returned within a fortnight.

The comment was made that she was a model patient, worked well under supervision, and apart from occasional outbursts of temper gave no trouble. Her mother, who was living openly with a man, had become interested in her again. Attempts had been made to place the patient in the country. An uncle, who had been approached, realized that there were sufficient immoral women in his town without adding to their numbers. The patient was a victim of her environment. In any remote country area her lack of intelligence would not be such a handicap as it was in the city; she would be likely to marry, settle down, have children and be contented. In the city she was a confirmed delinquent, was unlikely to improve, and seemed doomed to spend the rest of her life in mental hospitals.

Sexual Delinquency.

The next patient shown was a male, aged thirty years, who had been admitted to hospital on November 28, 1930;

his intelligence quotient was 47%. He had done poorly at school, which he left at the age of fourteen years. On leaving school he had worked for some time as a labourer, until his admission to the hospital at the age of fifteen years. It was stated at that time that he had attempted indecent assault against his mother; this had recently been denied. Since his admission to hospital the patient had been an excellent ward worker and a good footballer and cricketer, and had been interested in his surroundings. However, he had masturbated frequently, perhaps every day, and had also shown active and overt homosexual tendencies which necessitated his sleeping in a single room. His relatives had demanded his discharge from hospital, but this had been refused.

The final patient shown was a female, aged twenty years, whose intelligence quotient was 78%. She had had little schooling, but was said to have passed the primary final examination. She had run away from home at the age of thirteen, and was taken over by the Child Welfare Department. From June 17, 1938, to November 2, 1938, she was in the Mental Hospital, Morriset, but she was then transferred to the Mental Hospital, Rydalmere, as she was suspected of sexual delinquency with male patients. At Rydalmere she was a good worker, but had fits of petulance, when she was violent and resistive, requiring seclusion. She was allowed out on leave on June 12, 1940, but was returned on July 20; her guardian said that she was prone to tell falsehoods, and had been frequenting military camps, returning home at all hours; she proved to be pregnant.

The comment was made that since her return the patient had been an excellent worker, and her temper tantrums had been much less frequent and much milder than before. In many respects she had been more reliable than a nurse during the staff shortage.

VICTORIAN BRANCH NEWS.

THE following books have been added to the library of the Medical Society of Victoria (the Victorian Branch of the British Medical Association):

Agriculture.—C. Alma Baker: "The Labouring Earth", 1942. Sir Albert Howard: "An Agricultural Testament", 1944.

Allergy.—S. L. Feinberg: "Allergy in Practice", 1944.

Anæsthesia.—J. Ross Mackenzie: "Practical Anæsthetics", 1944.

Anatomy.—E. B. Jamieson: "Illustrations of Regional Anatomy", Fifth Edition, Sections 1-7. James Whillis: "Elementary Anatomy and Physiology", Second Edition, 1944.

Bacteriology.—R. R. Kudd: "Manual of Human Protozoa", First Edition, 1944. L. E. H. Whitby: "Medical Bacteriology", Fourth Edition, 1944.

Bones, Joints, Muscles.—J. G. Bonnin: "A Complete Outline of Fractures", 1941. C. W. Cutler: "The Hand: Its Disabilities and Diseases", 1942. Frederick Wood Jones: "The Foot: Structure and Function."

Dentistry.—Marzell Bronner and Max Bronner: "The Dental Surgeon's Handbook", 1944. W. J. Tuckfield: "Full Denture Technique", 1944.

Diagnosis.—R. L. Pullen (Editor): "Medical Diagnosis", 1944.

Digestive System.—B. P. Babkin: "Secretory Mechanism of the Digestive Glands." Martin E. Rehfuss: "Indigestion: Its Diagnosis and Management." Samuel Weiss: "Clinical Lectures on the Gallbladder and Bile Ducts", 1944. Stewart Wolff and H. S. Wolf: "Human Gastric Function."

Embryology.—W. J. Hamilton, J. D. Boyd and H. W. Mossman: "Human Embryology", 1945.

Endocrinology.—Jacob Hoffman: "Female Endocrinology", 1944.

Epidemiology.—J. S. Simmons *et alii.*: "Global Epidemiology", Volume I, 1944.

First Aid.—Lois Oakes: "Illustrations of Bandaging and First Aid", 1944.

Food, Dietetics, Nutrition.—J. S. McLester: "Nutrition and Diet in Health and Disease", 1944.

Genito-Urinary System.—Edward Weiss: "Practical Talks on Kidney Disease."

Heart and Circulatory System.—E. A. Barclay and K. J. Franklin: "The Fœtal Circulation." James B. Herrick: "A Short History of Cardiology." S. A. Levine: "Clinical Heart Disease", Third Edition, 1945. Arthur M. Master: "The Electrocardiogram and X-Ray Configuration of the Heart." A. Piney and Stanley Wyard: "Clinical Atlas of Blood Diseases", Sixth Edition, 1945. Hugo Roesler: "Clinical Roentgenology of the Cardiovascular System", 1943. David Harley: "Medico-Legal Blood Group Determination", 1943. A. Piney and P. J. L. Hamilton: "Sternal Puncture", 1943.

Hygiene: Personal and Public.—G. S. Parkinson: "Synopsis of Hygiene", Eighth Edition, 1944. Harvey Sutton: "Lectures on Preventive Medicine", 1944.

Industrial Medicine and Hygiene.—W. M. Gafafer (Editor): "Manual of Industrial Hygiene." F. J. Wampler (Editor): "Industrial Medicine", 1943.

Internal Medicine.—J. Murray Steele (Editor): "Advances in Internal Medicine", Volume I, 1942. Rudolf A. Stern: "Trauma in Internal Disease", 1945.

Medical Jurisprudence, Toxicology et cetera.—David Harley: "Medico-Legal Blood Group Determination", 1943. Sydney Smith: "Forensic Medicine", Eighth Edition, 1945.

Practice of Medicine.—R. S. Allison: "Sea Diseases." Beaumont and Dodds: "Recent Advances in Medicine", Eleventh Edition, 1944. "British Encyclopædia of Medical Practice", Volumes I-XII, 1939. Basil Graves: "Rational Medicine", 1944. H. H. Jordan: "Workmen's Compensation and the Physician", 1941. "Medical Annual", 1944. E. J. Stieglitz (Editor): "Geriatric Medicine", 1943.

Military Medicine.—Sir Arthur Hurst: "Medical Diseases of War", 1940.

Miscellaneous.—Janet Doe (Editor): "A Handbook of Medical Library Practice." Adolph Regli: "The Mayos—Pioneers in Medicine." Max. Thorek: "A Surgeon's World." A. E. Williams: "Barnado of Stepney", Frederick Wood Jones: "Habit and Heritage." Frederick Wood Jones: "Design and Purpose."

Neurology and Psychiatry.—Ruth Bochner: "The Clinical Application of the Rorschach Test", 1942. W. R. Brain and E. B. Strauss: "Recent Advances in Neurology", Fifth Edition, 1945. S. A. Kinnier-Wilson: "Neurology", Volumes I and II, 1944. Andre Ombredane: *"L'inhibition et la facilitation dans le système nerveaux central et périphérique."* E. A. Spiegel and I. Sinner: "Neurology of Eye, Ear, Nose and Throat", 1944. A. F. Tredgold: "Manual of Psychological Medicine." Edward Weiss and O. S. English: "Psychosomatic Medicine", 1943. B. Dattner: "The Managament of Neurosyphilis", 1944.

Obstetrics and Gynæcology.—E. A. Barclay and K. J. Franklin: "The Fœtal Circulation", 1944. Francis J. Browne: "Antenatal and Post-Natal Care", Fifth Edition, 1944. H. S. Crossen and R. J. Crossen: "Diseases of Women", Ninth Edition, 1941. A. H. Curtis: "Textbook of Gynæcology", Fourth Edition, 1944. Minnie Randell: "Training for Childbirth", Third Edition.

Ophthalmology.—John G. Bellows: "Cataract and Anomalies of the Lens", 1944. Charles May and Claud Worth: "Manual of Diseases of the Eye", 1944.

Oto-Rhino-Laryngology.—A. R. Hollender: "Office Treatment of Ear, Nose and Throat", 1943.

Pathology.—J. Martin Beattie and W. E. C. Dickson: "Textbook of Pathology", Fourth Edition, 1943. Wiley D. Forbus: "Reaction to Injury", 1943. R. F. Ogilvie: "Pathological Histology", Second Edition, 1943.

Pædiatrics.—A. H. Bowley: "Natural Development of the Child", Second Edition, 1943. Marion E. Breckenridge and E. Lee Vincent: "Child Development", 1943. W. C. Davison: "The Compleat Pediatrician", Fourth Edition, 1943. A. G. De Sanctis (Editor): "Advances in Pediatrics", Volume I, 1942.

Physiology.—Chalmers L. Gemmill: "Physiology in Aviation", 1943. James Whillis: "Elementary Anatomy and Physiology", Second Edition, 1944.

Radium and Röntgen Rays.—Arthur M. Master: "The Electrocardiogram and X-Ray Configuration of the Heart." Hugo Roesler: "Clinical Roentgenology of the Cardiovascular System", 1943.

Respiratory System.—Milton B. Rosenblatt: "Bronchiectasis."

Social Medicine.—Bureau of Medical Economics: "Medical Care in the United States", 1939. W. B. Doherty and D. D. Runes: "Rehabilitation of the War Injured", 1943. Sir Humphrey Rolleston (Editor): "Practitioner Booklet: After-Care and Rehabilitation", 1943. O. F. G. Smith: "Rehabilitation, Re-education and Remedial Exercises", 1943.

Surgery.—Hamilton Bailey: "Demonstrations of Physical Signs in Clinical Surgery", Ninth Edition, 1944. Hamilton Bailey (Editor): "Pye's Surgical Handicraft", Fourteenth

Edition, 1944. Harold Burrows: "Surgical Instruments and Appliances Used in Operations", Eleventh Edition, 1942. D. N. Matthews: "The Surgery of Repair", 1943. National Research Council: "Military Surgical Manuals", Volumes I to VI, 1942. Thomas G. Orr: "Operations of General Surgery", 1945. C. A. Pannett: "Surgery: Textbook for Students", 1944. James E. M. Thomson (Editor): "The American Academy of Orthopædic Surgeons: Lectures on Reconstruction Surgery", 1944. G. Grey Turner (Editor): "Carson's Modern Operative Surgery", Volumes I and II, Third Edition, 1943.

Therapeutics, Materia Medica, Pharmacology.—G. G. Lewis (compiled by): "The Ophthalmic Formulary", 1942. Frederick C. Smith: "Sulfonamide Therapy in Medical Practice", 1944.

Tuberculosis.—James Maxwell: "Care of Tuberculosis in the Home", 1943. Mervine C. Myerson: "Textbook of Tuberculosis of the Ear, Nose and Throat", 1944. T. N. Rafferty: "Artificial Pneumothorax in Pulmonary Tuberculosis", 1944.

Venereal Disease.—B. Dattner: "The Management of Neurosyphilis", 1944. Joseph Earle Moore: "Modern Treatment of Syphilis", Second Edition, 1941.

Special Correspondence.

NEW ZEALAND LETTER.

(By OUR SPECIAL CORRESPONDENT.)

Report on Referendum to Members of the British Medical Association Regarding Medical Benefits under Social Security.

FOR a long time, since the inauguration of medical benefits, there has been criticism of the scheme within the profession, and a desire to bring order out of the confusion arising from the different methods available to a practitioner of rendering the service. A *questionnaire* was sent out by the British Medical Association towards the end of 1944, and the results are published in the Supplement to *The New Zealand Medical Journal*, October, 1945. The highlights are as follow, with preliminary explanation.

Five methods of collecting for service are allowed by the Government—salary in special areas, capitation (the initial scheme adopted only by a few doctors), token payment by patient, "fee for service" and "refund". For practical purposes the last two are the most important. In the fee for service, which the Government intended to be the standard method, and which has been officially disapproved by the British Medical Association, the patient signs a chit that service has been rendered on specified date or dates, and the doctor sends in a heap of chits at intervals, and gets a cheque paying for the lot. In "refund", the method officially approved by the British Medical Association as being nearest to former conditions, the patient is billed and pays as before at whatever the doctor charges—usually basically 10s. 6d. On the receipt is specified the number of services with dates, and this receipt is a valid claim at the post office for the basic 7s. 6d. per service.

Twice as many general practitioners actually conduct their practices under the refund as under the fee-for-service method, but when asked to express their preference the ratio falls from 2:1 to 1·3:1. Incidentally there is a small increase in preference (as against fact) for capitation, and a rather marked one for salary.

A strong preference for "group practice" (meaning an association of medical practitioners for the pooling of knowledge and activity) appeared—610 *pro* and 155 *con*—in spite of the fact that no fully developed example of group practice by general practitioners is to be seen in the country. In this vote the specialist section was that most against group practice, and the general practitioners next, though there were 254 *pro* and 85 *con*.

Fairly definite answers as to the kind of group practice desired were forthcoming. A total of 504 favoured its voluntary establishment (237 against), whereas 575 were against rate or State aid, with 203 for it.

The most decisive answer of all came to the question: "Are you in favour of the New Zealand Branch of the British Medical Association resuming discussions with the Government?" Six hundred and fifty said "Yes" and 91 said "No".

First Post-War British Medical Association Conference in New Zealand.

The implications of these answers which show marked deviations from official British Medical Association doctrine will doubtless be discussed at the first post-war national British Medical Association conference, which is to be held in Auckland on February 12 to 15, 1946, under the presidency of Mr. A. Eisdell Moore, F.R.C.S. Sir Stanton Hicks, Professor of Physiology, Adelaide, will be present and speak. A full programme of scientific business and social affairs is being arranged.

Squadron Leader G. R. Lee, a layman, has just been appointed General Secretary to the Branch.

Annals of the Otago Medical School.

"Annals of the Otago Medical School", by Emeritus Professor of Medicine Dr. D. W. Carmalt-Jones, was published recently by A. H. and A. W. Reed and Company, 182, Wakefield Street, Wellington, at £1 1s. This book will prove of great interest to all friends and alumni of the school. The story is well, if calmly, told, and is one of small beginnings, enormous difficulties and great achievements.

Correspondence.

THE SEEDS OF MACROZAMIA DENISONI.

SIR: In a recent letter from Dr. W. H. Hoffmann, M.D., Director in the Finlay Institute, Habana, he has drawn my attention to the poisonous character of the seeds of *Macrozamia denisoni*. Quite by accident he gave one or two to a tame jutia (*Jutia conga*, a Cuban rodent reared in the laboratory). The rodent readily ate the seeds, but soon became depressed and died on the sixth day. At autopsy the stomach was found injected.

A guinea-pig was then tried. The first day it did not touch the nut, but on reduced food (some grass only) it ate some seed, two ordinary beans in size, and on the third day another small amount equal to one bean. On the fourth day it died. The stomach on autopsy was much dilated, the mucosa much injected, showing extensive hæmorrhages also visible on the serosa. Convincing evidence was thus given of death due to an acute poisoning, perhaps from a poison of the hydrocyanic acid group often found in other poisonous seeds, for example, almonds, cherries, though not in toxic quantities.

Macrozamia seeds are attractive in appearance and may be used by children in play. The risk is, as Dr. Hoffman points out, that the children may eat some, and, as the symptoms of intoxication will appear several days or even a week later, the real cause of the fatality may easily remain unsuspected.

Palm seeds such as those of the Royal Palm, *Oreodoxa regia*, known as the "Palma real" in Cuba, are an excellent food for pigs. This majestic palm, which may attain the height of fifty metres, is found in India, Ceylon, Indo-China and elsewhere in the tropics and produces enormous masses of small round seeds not bigger than a peppercorn.

Dr. R. Elliott Murray supplies me with the following Australian data. The macrozamias are not true palms, but cycads. The three Australian genera of Cycadaceæ contain poisonous species. Some of Captain Cook's men suffered (1770) and some of his pigs died as the result of eating nuts of *Cycas media*.

Nuts of macrozamia are noted as causing poisoning in man during the explorations of Grey (Western Australia, 1837) and Leichhardt (Queensland, 1847). Aborigines, however, eat the nuts, but only after very careful washing and roasting. In some species the leaves as well as the nuts contain the poisonous principle.

From time to time serious losses of cattle and sheep have resulted. The symptoms are either acute with dysentery, anæmia and gastro-enteritis, or chronic with partial paralysis of the hind limbs, resulting in a wobbly gait. This has been confirmed by the Poison Plants Committee of New South Wales in both cattle and sheep.

The poisonous principle of *Macrozamia spiralis*, the so-called "Burrawang palm", is being studied by Joyce Cooper. From an aqueous extract poisonous by oral administration to sheep she obtained (1940) crystals of macrozamin ($C_6H_{11}O_5N$). Ordinary hydrolysis with acids gives no HCN, but by hydrolysis with NaHO dilute, HCN is obtained (*Journal and Proceedings of the Royal Society of New South Wales*, Volume LXXIV, 1940).

The change into cyanide does not occur with the seeds of *Macrozamia spiralis* on eating, even though it readily occurs in a test tube (Finnemore).

Macrozamia denisoni is found on the coast in New South Wales and Queensland. Three boys were poisoned by it at Springsure in Queensland, 1899.

The danger alluded to by Dr. Hoffmann is definitely real.

Yours, etc.,

HARVEY SUTTON.

University of Sydney,
August 24, 1945.

GENERAL PRACTITIONERS AND SPECIALISTS.

SIR: There is an interesting letter from Alfred Cox in the *British Medical Journal* of June 9, 1945. He tells the story of his visit to Canada in 1924, when he called on a clergyman's wife who had been in Canada only a few years. This lady had found it practically impossible to get a family doctor as she understood the term. Her doctor informed her, when she was to have a baby, that he did not do midwifery. She had, therefore, to be attended by another doctor in hospital. Subsequently the infant developed an infantile malady and her doctor said that she must call in a children's specialist. The complaint turned out to be measles. General practitioners appeared to be an almost extinct species in Canadian cities. The same tendency, says the writer, exists in South Africa, where the proportion of specialists is very high.

As an army regimental medical officer, I have seen innumerable cases referred for specialist opinion who have no right to be. The regimental medical officer, to save himself the trouble of taking a decent case history and making a clinical examination, has passed on the case to the hospital specialist. Undoubtedly such practice is engendered in the regimental medical officer during his period as hospital resident medical officer when it is so easy to refer a case to the appropriate specialist or department. I personally give short shrift to the serviceman who walks into my regimental aid post, and, before even stating his complaint, asks to be sent for an X-ray examination or for specialist opinion. But, undoubtedly, army regimental medical officers have fostered such an attitude of mind amongst servicemen.

General practitioners in this country have enjoyed a remarkably high reputation for competence and versatility. If, however, the rising generation of general practitioners is not to became a very second-rate unit of the profession, it will have to take considerably more initiative and responsibility.

Vividly I remember the words of my Edinburgh teacher in medicine: "Take a careful case history. Then use the clinical methods you have been taught, using your eyes, your ears, your hands and fingers, and, not least, your nose. ["What is the first thing you do when examining a specimen of urine?" he would ask. His face would light up with pleasure and approbation if you gave him the answer he wanted, namely: "Smell it!"] Then, gentlemen, and not until then, make your requests for special investigations—X rays, laboratory tests and so on."

Such counsel might have been given by Sir James Mackenzie himself, but it is only too easy to forget it in these days of streamlined laboratory services.

Yours, etc.,

Ex-G.P.

September 18, 1945.

A MASS SURVEY OF CHESTS IN THE COUNTRY.

SIR: The Anti-Tuberculosis Association is contemplating an extension of its work from its city clinic to a mass survey of chests in the country and is asking for the supply of travelling X-ray mobile unit and a pathological unit.

I think this Association has little knowledge of the cost of such equipment and of its upkeep. Two separate units would be necessary. One truck would be needed for the X-ray unit (which would have an overall length of about twelve feet) and a dark room and a separate one for the pathological laboratory. Few country towns could supply the necessary current, and a large ten to fifteen kilowat generator driven by a petrol engine would have to be added, as an additional trailer unit. A generator driven by the car engine would cause vibration, and I doubt if it could give sufficient current. The X-ray transformer would have to be specially constructed, as the casing of the ordinary tank would not stand up to the rough roads which would be traversed. Lighting could be supplied from twelve-volt batteries and some type of refrigerating unit would be necessary to keep constant developing temperatures. The cost of such an outfit would run close to £10,000, and the staff necessary would be large—say, two technicians (one an expert electrician), a dark room attendant, a driver and a clerk, and possibly a nurse.

It will be seen that this is a costly business and could not be run without extensive government subsidy. Taken in all, I think it would be better for the Association to devote its funds to its city work and leave the mass chest survey to that financially starved section of the Health Department—the Tuberculosis Division.

Yours, etc.,

J. G. EDWARDS.

Craignish,
185, Macquarie Street,
Sydney.
September 21, 1944.

THE PROTECTION OF PRACTICES FUND OF THE EASTERN SUBURBS MEDICAL ASSOCIATION, NEW SOUTH WALES.

SIR: The Committee of the Eastern Suburbs Medical Association craves space in your columns for the report of the financial transactions of their "Protection of Practices" Fund. You kindly published the report of the first year's activities in 1940, and this further report is of the succeeding years to June 30, 1945.

PROTECTION OF PRACTICES SCHEME.

War Emergency Account.

A Summary of the Financial Transactions from July 1, 1941, to June 30, 1945.

July 1, 1941—	£ s. d.	£ s. d.
Credit balance Commonwealth Savings Bank of Australia, Bondi Junction Branch, S384 ..		355 4 6
Interest credited from July 1, 1941, to June 30, 1945	43 8 9	
Contributions received from July 1, 1941, to June 30, 1945	15,881 2 4	
		15,924 11 1
		16,279 15 7
Less payments to beneficiaries from July 1, 1941, to June 30, 1945		15,841 0 8
		438 14 11
Less sundry payments from July 1, 1941, to June 30, 1945		23 4 11
Credit bank balance as at June 30, 1945		£415 10 0

The noteworthy features of this fund are that a small group of public spirited men have contributed the amount of £19,450 over the space of five years for the security of those practitioners who left their practices. The total number of contributors was 62. The maximum number of beneficiaries at the one time was 21.

Another extraordinary point is that in the management of this large sum of money a total outlay of less than £50 has been necessary for incidental expenses.

For the Committee of the Eastern Suburbs Medical Association.

Yours, etc.,

C. M. BURNS,
Honorary Secretary.

154, Hopetoun Avenue,
Vaucluse,
November 20, 1945.

A CASE OF INTRACTABLE CONSTIPATION.

SIR: Today a patient of mine died at the age of seventy-eight years as the result of a carcinoma of the stomach. This, of course, is nothing out of the ordinary, but the fact that she had not had a bowel action for five and a half

weeks to me seemed most extraordinary, and it is because of this I write to your journal to publish same and to learn if any of your readers have had any similar case, or heard of any case that could beat this record. Her last bowel action was on the night of August 30. She had a good deal of vomiting and could only take liquids. Laxatives, purgatives and cathartics were of no avail, and enemata were merely returned without any result. During the whole of the above period the patient was perfectly comfortable. Her abdomen was soft and flat; the only distension present was two days before she died, and this was due to a full bladder which contained thirty ounces of urine obtained after catheterization. Her mental condition was good until a few hours before death. She was very emaciated. Her skin and subcutaneous tissues had a peculiar wrinkled and putty-like appearance. If pinched, the elevated tissues would remain in this glued-like state for hours. I have no reason to doubt the truth of the statement with regard to this marathon constipated state, as told to me by members of the family and the trained nurse who attended this patient. Not at any time was she in any pain, nor was there any reason to worry about the ineffectiveness of all methods used to obtain an evacuation. I felt that this freak of medical pathology was worth reporting.

Yours, etc.,

L. JOEL.

102, North Road,
Newport,
Victoria.
October 7, 1945.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 224 and 229, of November 22 and 29, 1945.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Promotion.—Surgeon Lieutenant Samuel Edward Lees Stening is promoted to the rank of Surgeon Lieutenant-Commander, dated 21st September, 1945.

Appointment.—Thomas Marden Moore is appointed Surgeon Lieutenant (D), dated 17th October, 1945.

Royal Australian Naval Volunteer Reserve.

Termination of Appointment.—The appointment of Robert Ewing Scott Charlton as Surgeon Lieutenant is terminated, dated 3rd September, 1945.

Promotions.—Surgeon Lieutenant-Commanders Reginald Francis Matters (Acting Surgeon Commander) and Donald Dunbar Coutts, D.S.O., are promoted to the rank of Surgeon Commander, dated 15th August, 1945.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The appointment of Temporary Flight Lieutenant G. J. Ramsay (256308) is terminated on demobilization with effect from 12th September, 1945.

The appointment of Temporary Squadron Leader R. C. Angove (281217) is terminated on having completed long service with effect from 3rd September, 1945.

The appointments of the following officers are terminated on demobilization with effect from the dates indicated: Temporary Squadron Leader J. M. Alexander (261669), 12th September, 1945, Temporary Flight Lieutenant J. E. Carroll (267573), 4th September, 1945.

Reserve: Medical Branch.

The following Temporary Squadron Leaders are transferred from the Active List with effect from the dates indicated: B. Epstein (261714), 22nd August, 1945, B. M. L. Dunstone (281654), 28th August, 1945, E. G. Strahan (251778), 4th September, 1945.—(Ex. Min. No. 271—Approved 28th November, 1945.)

Temporary Squadron Leader R. V. Pridmore (281260) is transferred from the Active List with effect from 25th August, 1945.—(Ex. Min. No. 272—Approved 28th November, 1945.)

The Royal Australasian College of Physicians.

ADMISSION OF FELLOWS AND MEMBERS.

AT a meeting of the general body of Fellows of the Royal Australasian College of Physicians, held at Sydney on September 27, 1945, Dr. Walter Gilmour and Dr. Michael Gerbert Watt, of New Zealand, were elected and admitted to Fellowship of the College.

The following candidates, who were successful at an examination for membership of the College held at Sydney in September, 1945, were admitted to membership at a meeting of the Council of the College held on September 27, 1945: Surgeon Lieutenant J. M. Alexander, R.N.V.R., of Kent, England; Major M. L. Edwards, Dr. M. J. Heseltine, Major J. V. Latham, Captain M. G. Lusby, Dr. J. S. Paton, Major R. B. Perkins, and Squadron-Leader J. N. Sevier, of New South Wales; Major B. L. Hellings and Dr. H. N. B. Wettenhall, of Victoria; Major G. R. Kurrle, of Queensland.

Obituary.

FREDERICK JOHN BRIDGES.

WE are indebted to Dr. M. S. S. Earlam for the following appreciation of the late Dr. Frederick John Bridges.

Although, as a result of the illness which was ultimately to prove fatal, Frederick Bridges had not been in practice for several years, his death came as a shock to his many friends.

Graduating in 1911, he was appointed as honorary assistant physician to the Royal Prince Alfred Hospital, and later as honorary physician to the Royal North Shore Hospital. He resigned from the former position in 1928, and from Royal North Shore Hospital in 1936, when he relinquished practice. From then until his death he occupied himself in the main with literary activities—often but not necessarily with a medical flavour—general journalism, book reviewing, sub-editing of medical and scientific publications and general editorial work. He also did a considerable amount of medical broadcasting, and for a period of seven years had a large and regular audience as "Doctor Frederick", of Station 2UW.

Frederick was one of those personalities of medicine who are all too rare. He was tremendously well informed on music, history, art and literature, and was himself possessed of a style and wit not often encountered. One remembers being associated in resident days with him in the Anti-tuberculosis Dispensary at the Royal Prince Alfred Hospital when it was in charge of the late Sinclair Gillies—a happy association indeed, so much so that on one occasion Frederick's chortling and whistling while occupied with his patients in an adjacent room prompted Sinclair Gillies to exclaim: "What the devil does that fellow Bridges think he's doing out there—nesting?" In his company one never had a dull moment. "Ah! Mrs. Jones", he said once in the course of a routine oral examination, "what have we here? A golden crown. Never forget, Mrs. Jones, uneasy lies the head that wears a crown." And so on *ad infinitum*. For years after it occurred the story was still being told how, when he was a junior resident anæsthetizing for a very senior gynæcologist who liked his patients very deeply anæsthetized, the latter complained that the patient's abdomen was moving, on which Frederick immediately offered up fervent and heartfelt thanks to what he felt were the appropriate quarters. If he ever had dull moments he did not allow his friends to share them. He was a unique and lovable character and the profession is the poorer for his passing.

DR. KEMPSON MADDOX writes: Frederick John Bridges ("Captain Bridges" to his medical intimates) was an unusual doctor. His gifts were infrequently on display, but to those of us who knew him well were rare and delightful. His undoubted ability as a physician was such that, had not bad health fallen upon him in the hour of opportunity, he would unquestionably have attained high medical fame in both consulting room and hospital. His lecturing and tutorials would have given enduring glee to generations of students. His delicious capacity as a raconteur, critic and writer was of a rare, lamentably rare, quality. His literary and musical knowledge would have carried him far in either of these fields, even against the best professional competition. It is

unfortunate that many of his medical writings and sayings were to be found only in some "popular" form. After terminating general practice, the irritations of which were quite incompatible with his temperament, he at last found leisure to enjoy his greater interests which were closely shared by his wife. Unfortunately, too, he became somewhat of a recluse, so that his old friends saw him far too rarely. A generous friend, a delightful companion, and a brilliant essayist whom they cannot readily replace.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Adams, David Joseph Monk, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Andersen, Neville Arthur, M.B., B.S., 1945 (Univ. Sydney), Marrickville District Hospital, Marrickville.
Anderson, Donald Edmund, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Barbour, John Gordon, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Bell, Betty Jeanette, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Bolliger, Walter, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Bruce-Smith, Bruce, M.B., B.S., 1945 (Univ. Sydney), Royal Hobart Hospital, Hobart, Tasmania.
Brunton, Lawrence Jackson, M.B., 1945 (Univ. Sydney), Launceston Hospital, Launceston, Tasmania.
Bunn, Terence Aubrey, M.B., B.S., 1945 (Univ. Sydney), Mater Misericordiæ Hospital, Waratah.
Burton-Bradley, Burton Gyrth, M.B., 1945 (Univ. Sydney), Western Suburbs Hospital, Croydon.
Butler, John Ferdinand, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Champion, Elizabeth Patterson, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Chancellor, Alan Harold Bennet, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Cleland, Kenneth Wollaston, M.B., B.S., 1945 (Univ. Sydney), Goulburn District Hospital, Goulburn.
Cockburn, Keith Gordon, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Collins, Ian Stuart, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Courtenay, Kevin Hector, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Cuthbert, Antony Bevan, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
d'Apice, Mary Dorothy, M.B., B.S., 1945 (Univ. Sydney), Saint George District Hospital, Kogarah.
Davis, Neville Coleman, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Dawson, Lionel Adrian, M.B., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Doyle, Evelyn Mary, M.B., B.S., 1945 (Univ. Sydney), Perth General Hospital, Perth, Western Australia.
Dunn, David Brian, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Elphinstone, Keith Raymond, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Failes, John Watson, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Farrar, Frank Martindale, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Fraser, Allan Arthur, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Frost, Thomas James, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Gerdes, William Thomas, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Giblin, William Eric, M.B., B.S., 1945 (Univ. Sydney), Mater Misericordiæ Hospital, North Sydney.
Goldie, John Ernest Dunlop, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Grant, John Macdonald Falconar, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Grant, Peter John Falconar, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Green, Robert Musgrave, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Grunseit, Ferry, M.B., B.S., 1945 (Univ. Sydney), Royal Hobart Hospital, Hobart, Tasmania.
Hagen, Paul Beo, M.B., B.S., 1945 (Univ. Sydney), Balmain and District Hospital, Balmain.
Harrison-Potter, Frances Del, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Hawker, Ross Wilson, M.B., B.S., 1945 (Univ. Sydney), Royal Hobart Hospital, Hobart, Tasmania.
Herlihy, William Francis, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Hicks, Zoe Isobel, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Higham, Robert Harvey, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Hilliar, Bruce Stanley, M.B., B.S., 1945 (Univ. Sydney), Newcastle Hospital, Newcastle.
Hirst, Edmond, M.B., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Hooper, Francis Maxwell, M.B., B.S., 1945 (Univ. Sydney), Saint George District Hospital, Kogarah.
Horsley, Roberta Gertrude, M.B., B.S., 1945 (Univ. Sydney), Manly District Hospital, Manly.
Howe, Rene Edgar, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Howell, David John, M.B., B.S., 1945 (Univ. Sydney), Western Suburbs Hospital, Croydon.
Hunt, Thelma May, M.B., B.S., 1945 (Univ. Sydney), Saint George District Hospital, Kogarah.
Hunter, William Fyfe, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Indyk, Jack Solomon, M.B., B.S., 1945 (Univ. Sydney), Royal Hobart Hospital, Hobart, Tasmania.
Jackson, Murray Archibald, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
King, Errol Edward Trevor, M.B., B.S., 1945 (Univ. Sydney), Saint George District Hospital, Kogarah.
Knight, Ronald Victor, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Lane, Melva Joyce, M.B., B.S., 1945 (Univ. Sydney), Saint George District Hospital, Kogarah.
Lang, William Robson, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Lawson, Deryk Willard, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Lehmann, Douglas Keith, M.B., B.S., 1945 (Univ. Sydney), Hornsby and District Hospital, Hornsby.
Letham, John Burnside, M.B., B.S., 1945 (Univ. Sydney), Brisbane General Hospital, Brisbane, Queensland.
Lofberg, Jack Valdamar, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Lyttle, Keith Wightman Purves, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.
Macartney, Joan Ellison, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
McCaffery, Louis Henry, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
McCarthy, William Justin, M.B., 1945 (Univ. Sydney), Lewisham Hospital, Lewisham.
McDonald, Geoffrey Lance, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
McDowell, Moira Agnes, M.B., 1945 (Univ. Sydney), Brisbane General Hospital, Brisbane, Queensland.
Mackaness, George Bellamy, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
MacMillan, Kenneth Charles, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.
Manion, Newman Eugene, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Mansell, Hazel, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Manuel, Betty Luce, M.B., B.S., 1945 (Univ. Sydney), Rachel Forster Hospital for Women and Children, Redfern.
Manzie, Peter Podmore, M.B., B.S., 1945 (Univ. Sydney), Newcastle Hospital, Newcastle.
Martin, James Matthew, M.B., B.S., 1945 (Univ. Sydney), Mater Misericordiæ Hospital, North Sydney.
Matchett, Victor Lewin, M.B., B.S., 1945 (Univ. Sydney), Fremantle Hospital, Fremantle, Western Australia.
Mead, Kevin William, M.B., B.S., 1945 (Univ. Sydney), Brisbane General Hospital, Brisbane, Queensland.
Merton, Thomas Allenby, M.B., 1945 (Univ. Sydney), Perth General Hospital, Perth, Western Australia.
Millingen, Keith Samual, M.B., B.S., 1945 (Univ. Sydney), Sydney Hospital, Sydney.

Medical Appointments.

Dr. T. G. C. Retallick has been appointed Medical Superintendent, Mental Hospital, Sunbury, Victoria.

Dr. H. J. C. Edmonds has been appointed Medical Superintendent, Mental Hospital, Beechworth, Victoria.

Dr. S. Matthews has been appointed a member of the Pingelly Hospital Board, Western Australia, for a period of two years.

Dr. C. G. Semler has been appointed Temporary Resident Medical Officer of the Royal Adelaide Hospital, South Australia.

Dr. P. N. Grieve has been appointed Government Medical Officer at Liverpool, New South Wales.

Dr. T. Ambrose has been appointed Medical Officer of Health, Meekatharra Road Board, pursuant to the provisions of *The Health Act*, 1911-1944, of Western Australia.

Dr. George Shaw has been appointed Medical Health Officer for the Zeehan District, Zeehan Municipality, Tasmania.

Dr. W. D. Ackland-Horman, Dr. C. G. Semler and Dr. R. G. Plummer have been appointed registrars of the Royal Adelaide Hospital, Adelaide, South Australia.

Books Received.

"Introduction to Diseases of the Chest", by James Maxwell, M.D. (London), F.R.C.P. (London); Second Edition; 1945. London: Hodder and Stoughton Limited. 8¼" × 5¼", pp. 335, with many illustrations. Price: 12s. 6d. net.

"Hæmoglobin Levels in Great Britain in 1943 (with Observations upon Serum Protein Levels)", by the Committee on Hæmoglobin Surveys, Medical Research Council of the Privy Council, Special Report Series, Number 252; 1945. London: His Majesty's Stationery Office. 9¼" × 6", pp. 134. Price: 2s. net.

"Control of Pain in Childbirth", by Clifford B. Lull, M.D., F.A.C.S., and Robert A. Hingson, M.D., with an introduction by Norris W. Vaux, M.D.; Second Edition; 1945. Philadelphia: J. B. Lippincott Company. 9" × 6", pp. 376, with many illustrations. Price: 56s.

"Obstetric Management and Nursing", by Henry L. Woodward, M.D., and Bernice Gardner, R.N., with a section on home deliveries by William P. Gillespie, M.D., and also with a section on diseases of the newly born by Harold F. Downing, M.D.; Second Edition; 1944. Philadelphia: F. A. Davis Company. 8¼" × 5¾", pp. 772, with many illustrations. Price: 28s.

"Hospital Color and Decoration", by Raymond P. Sloan; 1944. Chicago: Physicians' Record Company. 9¼" × 7", pp. 272, with many illustrations. Price: 32s. 6d.

"Chronic Pulmonary Disease in South Wales Coalminers: III. Experimental Studies", Medical Research Council of the Privy Council, Special Report Series, Number 250; 1945. London: His Majesty's Stationery Office. 9¼" × 6", pp. 101, with many illustrations. Price: 5s. net.

"The Principles and Practice of Rectal Surgery", by William B. Gabriel, M.S. (London), F.R.C.S. (England); Third Edition; 1945. London: H. K. Lewis and Company Limited. 9¾" × 6", pp. 442, with many illustrations. Price: 45s. net.

"Modern Treatment Year Book, 1945: A Year Book of Diagnosis and Treatment for the General Practitioner", edited by Cecil P. G. Wakeley, C.B., D.Sc., F.R.C.S., F.R.S.E., F.A.C.S. London: The Medical Press and Circular. 8¼" × 5¼", pp. 308, with 60 illustrations, including 15 plates. Price: 15s.

"A History of Medicine", by Douglas Guthrie, M.D., F.R.C.S. (Edinburgh), F.R.S.E.; 1945. Edinburgh: Thomas Nelson and Sons Limited. 8¾" × 5¾", pp. 464, with 72 plates. Price: 30s. net.

"Handbook of Diagnosis and Treatment of Venereal Diseases", by A. E. W. McLachlan, M.B., Ch.B. (Edinburgh), D.P.H., F.R.S. (Edinburgh); Second Edition; 1945. Edinburgh: E. and S. Livingstone Limited. 7¼" × 5", pp. 379, with many illustrations, some in colour. Price: 15s. net, postage 7d.

"Aviation Neuro-Psychiatry", by R. N. Ironside, M.B. (Aberdeen), F.R.C.P. (London), and I. R. C. Batchelor, M.B. (Edinburgh); 1945. Edinburgh: E. and S. Livingstone Limited. 8¼" × 5¼", pp. 175. Price: 8s. 6d. net, postage 6d.

"Coast to Coast: Australian Stories, 1944", selected by Vance Palmer; 1945. Sydney: Angus and Robertson Limited. 7¼" × 5", pp. 264. Price: 7s. 6d.

"What People Are: A Study of Normal Young Men", by Clark W. Heath; 1945. Cambridge, Massachusetts: Harvard University Press. 8¼" × 5¼", pp. 157. Price: $2.00.

"The Medical Annual: A Year Book of Treatment and Practitioner's Index", edited by Sir Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P., and A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; 1945—sixty-third year. Bristol: John Wright and Sons Limited. 8¾" × 5¾", pp. 422. Price: 25s. net.

"Pulmonary Edema and Inflammation: An Analysis of Processes Involved in the Formation and Removal of Pulmonary Transudates and Exudates", by Cecil K. Drinker, M.D., D.Sc.; 1945. Cambridge, Massachusetts: Harvard University Press. 8¼" × 5¼", pp. 116, with many illustrations. Price: $2.50.

"Virus as Organism: Evolutionary and Ecological Aspects of Some Human Virus Diseases", by Frank MacFarlane Burnet, M.D., F.R.S.; 1945. Cambridge, Massachusetts: Harvard University Press. 8¼" × 5¼", pp. 144. Price: $2.00.

"Medical Jurisprudence and Toxicology", by John Glaister, J.P., D.Sc., M.D., F.R.S. (Ed.); Eighth Edition; 1945. Edinburgh: E. and S. Livingstone Limited. 8¼" × 5¼", pp. 703, with many illustrations, some in colour. Price: 30s. net.

"Essentials of Surgery for Dental Students", by J. Cosbie Ross, M.B., Ch.M. (Liverpool), F.R.C.S. (England); 1945. Edinburgh: E. and S. Livingstone Limited. 8¼" × 5¼", pp. 292, with many illustrations. Price: 20s. net.

"Pediatric X-Ray Diagnosis: A Textbook for Students and Practitioners of Pediatrics, Surgery and Radiology", by John Caffey, A.B., M.D.; 1945. Chicago: The Year Book Publishers, Incorporated. 10" × 6¾", pp. 860, with many illustrations. Price: $12.50.

Diary for the Month.

DEC. 26.—Victorian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All Public Health Department appointments.

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